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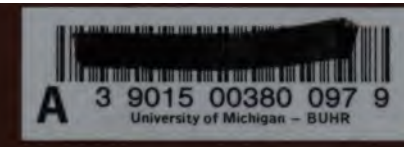
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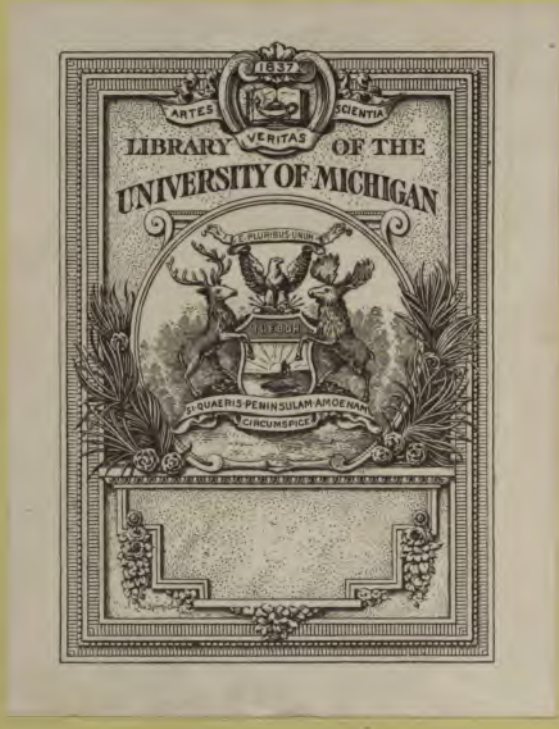
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20, HANOVER SQUARE, W.
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1902 ARMOUR, DONALD JOHN, M.B., 89, Harley Street, W. *C.C.* 2.
1885 ARMSTRONG, HENRY GEORGE, Wellington College, Berkshire.
1888 ARMSTRONG, HUGH, Tamworth, New South Wales.
O.M. ARNOTT, HENRY, The Rectory, 32, High Street, Beckenham, Kent. (C. 1871–5.) *Trans.* 3.
1903 ATKINSON, THOMAS REUELL, M.D., Chadwell Heath, Essex.
1894 ATTLEE, JOHN, M.D., B.C., 65, Grosvenor Street, W. *C.C.* 2.
1898 AVERY, LEONARD, 15, Chesterfield Street, Mayfair, W.
1905 BAILEY, WILLIAM HENRY, M.D., Featherstone Hall, Southall.
1880 BAKER, HENRY FRANCIS, 2, Mandeville Place, Manchester Square, W.

ELECTED

1895 BALDWIN, GERALD R., 166, Victoria Street, Melbourne, Australia. *Trans.* 1.

1883 BALLANCE, CHARLES ALFRED, M.B., M.S., 106, Harley Street, W. (C. 1894–6.) *Trans.* 3, *C.C.* 6.

1904 BALLANCE, H. A., 46, Prince of Wales Road, Norwich.

1888 BANHAM, HENRY FRENCH, M.D., Assington Vicarage, Colchester.

1868 BANTOCK, GEORGE GRANVILLE, M.D., 14, Upper Hamilton Terrace, N.W.

1876 BARKER, ARTHUR E. J., 87, Harley Street, W. (C. 1883–5, V.P. 1901–3.) *Trans.* 19, *C.C.* 14.

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service, India.

1894 BARLING, HARRY GILBERT, 87, Cornwall Street, Newhall Street, Birmingham. (C. 1901–3.) *Trans.* 3.

1875 BARLOW, SIR THOMAS, Bart., K.C.V.O., M.D. (*President*), 10, Wimpole Street, W. (C. 1880–2, 1891–4, S. 1888–90, V.P. 1895–8. P. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 11, *C.C.* 5.

1902 BARNARD, HAROLD L., 21, Wimpole Street, Cavendish Square, W. *Trans.* 2, *C.C.* 10.

1888 BARRS, A. G., M.D., 22, Park Place, Leeds.

1888 BARTON, EDWIN A., 56, Kensington Court, Kensington, W.

1891 BARTON, J. KINGSTON, 14, Ashburn Place, Courtfield Road, S.W. (C. 1901–4.)

1901 BARWELL, HAROLD SHUTTLEWORTH, 55, Wimpole Street, Cavendish Square, W.

O.M. BARWELL, RICHARD, 55, Wimpole Street, Cavendish Square, W. (C. 1872–5, V.P. 1877–9.) *Trans.* 21, *C.C.* 2.

O.M. **BASTIAN**, HENRY CHARLTON, M.D., F.R.S., 8A, Manchester Square, W. (C. 1876–8, V.P. 1891–3.) *Trans.* 7.

1896 BATTEN, FREDERICK E., M.D., 33, Harley Street, W. (C. 1903–6.) *Trans.* 5, *C.C.* 11.

1899 BATTEN, RAYNER DERRY, M.D., B.S., 33, Harley Street, W. *C.C.* 1.

1888 BATTERHAM, JOHN WILLIAMS, M.B., B.S., Bank House, Grand Parade, St. Leonard's-on-Sea.

1886 BATTLE, WILLIAM HENRY, 49, Harley Street, W. (C. 1899–1902.) *Trans.* 7, *C.C.* 19.

1868 BÄUMLER, CHRISTIAN G. H., M.D., The University, Freiburg i/Br., Baden, Germany. *Trans.* 4.

1898 BEDDARD, ARTHUR PHILIP, M.D., 44, Seymour Street, Portman Square, W.

1880 **BEEVOR**, CHARLES EDWARD, M.D., 135, Harley Street, W. (C. 1890–1.) *Trans.* 2, *C.C.* 10.

1901 BEEVOR, SIR HUGH REEVE, Bart., M.D., 17, Wimpole Street, Cavendish Square, W. *C.C.* 4.

ELECTED

1884 BENHAM, F. LUCAS, M.D., Campden House, nr. Exeter, Semaphore, South Australia. *Trans.* 4.

1883 BENHAM, ROBERT FITZROY, 11, Earl's Court Square, S.W.

1874 BENNETT, Sir WILLIAM HENRY, K.C.V.O., 1, Chesterfield Street, W. (C. 1889, 1892–4, S. 1890–1, V.P. 1895–8.) *Trans.* 4, *C.C.* 3.

1896 BENTHAM, EDWARD COOPER, 10, Princes Street, Cavendish Square, W.

1889 BENTLEY, ARTHUR J. M., M.D., Mena House, Pyramids, Cairo, Egypt.

1882 BERRY, FREDERICK HAYCRAFT, M.D., Watford, Herts.

1885 BERRY, JAMES, B.S., 21, Wimpole Street, W. (C. 1898–1901.) *Trans.* 2, *C.C.* 1.

1889 BIDWELL, LEONARD ARTHUR, 15, Upper Wimpole Street, W. (C. 1901–4.) *Trans.* 5, *C.C.* 1.

1882 BINDLEY, PHILIP HENRY, M.B. (Travelling).

1889 BISSHOPP, FRANCIS ROBERT BRYANT, M.A., M.D., B.S., Parham House, Tunbridge Wells.

1905 BLAKE, WILLIAM HENRY, M.D., Bedford Lodge, West Wickham, Kent.

1904 BOLTON, CHARLES, M.D., 16, Devonshire Street, Portland Place, W. *C.C.* 1.

1893 BOND, JAMES WILLIAM, M.D., 26, Harley Street, W. *C.C.* 2.

1898 BOSANQUET, WILLIAM CECIL, M.D., 117A, Harley Street, Cavendish Square, W.

1900 BOUSFIELD, ARTHUR, M.B., The Cheal, Hornsey Lane, N.

1899 BOWLAN, MARCUS MARWOOD, M.B., B.S., The Infirmary, Raine Street, St. George-in-the-East, E.

1883 BOWLBY, ANTHONY A., C.M.G., 24, Manchester Square, W. (C. 1891–3, 1904–7, S. 1901–4. V.-P. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 6, *C.C.* 4.

1883 Bowles, ROBERT LEAMON, M.D., Prior's Mesne, Lydney, Gloucestershire. (C. 1890–1.)

1896 BOX, CHARLES R., M.D., 2, Devonshire Place, Portland Place, W. (C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 3.

1889 BOYD, STANLEY, B.S., 134, Harley Street, W. (C. 1899–1902.) *Trans.* 1, *C.C.* 7.

1890 BRADFORD, JOHN ROSE, M.D., F.R.S., 8, Manchester Square, W. (C. 1900–1902.) *Trans.* 2.

1878 BRIDGES, ROBERT, M.B., M.A., The Manor House, Yattendon, Berkshire. *Trans.* 1.

1868 BRIGHT, GEORGE CHARLES, M.D., Cannes, Alpes Maritimes, France.

1887 BROCK, J. H. E., M.D., B.S., 77, Fellows Road, South Hampstead, N.W.

1894 BRODIE, C. GORDON, Fernhill, Wootton Bridge, Isle of Wight. *C.C.* 1.

1905 BROOK, WILLIAM FREDERICK, Longland House, Swansea. *C.C.* 1.

1898 Brook, WILLIAM HENRY BREFFIT, M.D., B.S., 8, Eastgate, Lincoln. (C. 1906–7. C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 4, *C.C.* 1.

ELECTED

1876 BROWNE, GEORGE BUCKSTON, 80, Wimpole Street, W. (C. 1891–3.) *Trans.* 3.

1904 BROWNE, H. S. D., M.B., B.C., 78, High Street, Winchester.

1887 BROWNE, OSWALD AUCHINLECK, M.A., M.D., 7, Upper Wimpole Street, W.

1883 BRUCE, JOHN MITCHELL, M.D., 23, Harley Street, W. (C. 1901–4.)

1905 BRYDONE, JAMES MEAR, M.B., B.C., 16, South Audley Street, Grosvenor Square, W.

1890 BUCKLAND, FRANCIS O., B.A., M.B., C.M., Brockenhurst, Hants. *Trans.* 1.

1901 BUCKNALL, THOMAS RUPERT HAMPDEN, M.S., 16, Devonshire Street, W. *C.C.* 1.

1891 BURGHARD, FREDERIC FRANÇOIS, M.D., M.S., 86, Harley Street, W. *Trans.* 1.

1879 BURTON, WILLIAM EDWARD, 24, Wimpole Street, W.

1900 ZUM BUSCH, JOSEF PAUL, M.D., Coventry House, South Place, Finsbury Pavement, E.C. *Trans.* 1, *C.C.* 1.

1881 BUTLIN, HENRY TRENTHAM, 82, Harley Street, W. (C. 1887–9, V.P. 1904-6.) *Trans.* 8, *C.C.* 1.

1884 BUXTON, DUDLEY WILMOT, M.D., B.S., 82, Mortimer Street, W.

1902 BUZZARD, EDWARD FARQUHAR, M.D., 33, Harley Street, W. *Trans.* 2.

O.M. BUZZARD, THOMAS, M.D., 74, Grosvenor Street, W. (S. 1870–2, C. 1873–6, V.P. 1880–1, P. 1895–7.) *Trans.* 15, *C.C.* 3.

1901 CADDY, ADRIAN, M.D., 2/2, Harington Street, Calcutta.

1891 CADDY, ARNOLD, 2/2, Harington Street, Calcutta.

1893 CALEY, HENRY ALBERT, M.D., 24, Upper Berkeley Street, W. *C.C.* 1.

1890 CALVERT, JAMES, M.D., 113, Harley Street, Cavendish Square, W. *Trans.* 2, *C.C.* 2.

1893 CAMPBELL, HARRY, M.D., 23, Wimpole Street, W., and Coniston Cottage, Fox Lane, Upper Norwood, S.E. (C. 1904-7.) *C.C.* 5.

1891 CARLESS, ALBERT, M.S., M.B., 6, Upper Wimpole Street, W. (C. 1905–7. C. Clin. Sect. Roy. Soc. Med., 1907– .)

1905 CARLING, E. ROCK, M.B., B.S., 38, New Cavendish Street, W.

1890 CARR, JOHN WALTER, M.D., 19, Cavendish Place, W. (C. 1903–6.) *Trans.* 3, *C.C.* 7.

1901 CARSON, HERBERT WILLIAM, 26, Welbeck Street, Cavendish Square, W. *Trans.* 1.

1901 Carwardine, THOMAS, M.S., 16, Victoria Square, Clifton, Bristol. *Trans.* 1.

1885 CATON, RICHARD, M.D., 86, Rodney Street, Liverpool. *Trans.* 3.

1890 CHAPMAN, CHARLES WILLIAM, M.D., 21, Weymouth Street, W. (C. 1900–3.) *Trans.* 3, *C.C.* 2.

1884 CHAPMAN, PAUL M., M.D., 1, St. John's Street, Hereford. *C.C.* 1.

ELECTED

1906 CHARLES, JOHN ROGER, M.D., 12, Victoria Square, Clifton, Bristol.

1893 CHARSLEY, ROBERT STEPHEN, "The Old Barn," Slough.

1885 CHEYNE, W. WATSON, C.B., M.B., C.M., F.R.S., 75, Harley Street, W. (C. 1894–6.) *Trans.* 2, *C.C.* 9.

1873 CHISHOLM, EDWIN, M.D., 44, Roslyn Gardens, Darlinghurst, Sydney, New South Wales.

O.M. **CHURCH**, Sir WILLIAM SELBY, Bart., K.C.B., M.D., 130, Harley Street, W. (C. 1874–6.)

1873 CHURTON, THOMAS, M.D., 35, Park Square, Leeds. (C. 1889–91.) *Trans.* 6.

O.M. CLAPTON, EDWARD, M.D., Towercroft, 41, Eltham Road, Lee, S.E. (C. 1872–4.) *Trans.* 1.

1874 CLARK, ANDREW, 71, Harley Street, W. *Trans.* 1.

1887 CLARK, FRANCIS WILLIAM, Hong-Kong.

1898 CLARKE, JAMES JACKSON, M.B., 18, Portland Place, W. *Trans.* 4, *C.C.* 11.

1885 Clarke, J. MICHELL, M.D., 28, Pembroke Road, Clifton, Bristol.

1885 CLARKE, WILLIAM BRUCE, M.B., 51, Harley Street, W. (C. 1894–7.) *Trans.* 2, *C.C.* 3.

1877 Clay, ROBERT HOGARTH, M.D., 4, Windsor Villas, Plymouth.

1877 **CLUTTON**, HENRY HUGH, M.A., 2, Portland Place, W. (C. 1885–7, V.-P. 1902–4, P. 1905–7.) *Trans.* 14, *C.C.* 4.

1898 COBB, ERNEST HERBERT, Belmont, Stevenage, Herts.

1893 COLE, ROBERT HENRY, M.D., Moorcroft, Hillingdon, Middlesex.

1882 COLLIER, HERBERT, M.D., 21, South Quay, Great Yarmouth, Norfolk.

1902 COLLINS, VICTOR EVELYN, M.B., Simons Town, Cape Colony.

1896 COLMAN, WALTER STACY, M.D., 9, Wimpole Street, W. (C. 1903–6.) *Trans.* 2, *C.C.* 3.

1882 COLQUHOUN, DANIEL, M.D., Dunedin, New Zealand.

1868 COOPER, FRANK W. (address uncommunicated).

1900 COOPER, HARRY, M.D., Fownhope, Surbiton Hill, Surrey.

1903 COOPER, PERCY ROBERT, M.D., "Glenthorn," The Downs, Bowdon, Cheshire.

1902 CORNER, EDRED MOSS, B.Sc., M.B., 37, Harley Street, W. *Trans.* 7, *C.C.* 7.

O.M. COUPER, JOHN, 80, Grosvenor Street, W. (C. 1874.)

1875 COUPLAND, SIDNEY, M.D., 16, Queen Anne Street, W. (S. 1882–4, C. 1885–7, V.-P. 1898–1901.) *Trans.* 5, *C.C.* 3.

1886 COUSINS, JOHN WARD, M.D., Riversdale, Kent Road, Southsea. *Trans.* 1.

1882 COXWELL, C. F., M.B., Brighton, Melbourne, Victoria. *Trans.* 2.

1897 CRAWFURD, RAYMOND H. P., M.D., 71, Harley Street, Cavendish Square, W. *C.C.* 3.

1879 CRIPPS, WILLIAM HARRISON, 2, Stratford Place, W. (C. 1886–8.) *Trans.* 3.

1872 CRITCHETT, Sir G. ANDERSON, 21, Harley Street, W.

ELECTED

1877 CROCKER, HENRY RADCLIFFE, M.D., 121, Harley Street, W. (C. 1884–5.) *Trans.* 16.

1896 CROOKE-LAWLESS, W. R., Surg.-Maj. A.M.S., M.D., Guards' Club, S.W.

1905 CUNNING, JOSEPH, M.B., B.S., 3, Upper Wimpole Street, W. *Trans.* 1.

1893 CURTIS, HENRY JONES, M.D., B.S., 58, Harley Street, W. *C.C.* 1.

1902 CUTHBERT, CHARLES FIRMIN, 2, Barton Street, Gloucester.

1872 DALBY, Sir WILLIAM BARTLETT, M.B., 18, Savile Row, W. (C. 1879–81.) *Trans.* 4.

1896 DALTON, NORMAN, M.D., 4, Mansfield Street, Portland Place, W. (C. 1904–7.) *Trans.* 4, *C.C.* 7.

1898 DARLEY-HARTLEY, WILLIAM, M.D., Seapoints, Cape Town, S. Africa.

1879 Davy, HENRY, M.D., 29, Southernhay, Exeter.

1889 DEAN, HENRY PERCY, M.B., 69, Harley Street, W.

1897 DEANE, JOHN HENRY, 50, Wallwood Road, Leytonstone, Essex.

1899 DE CHAZAL, EDMOND LUCIEN, M.D., Port Louis, Mauritius.

1903 DELBRUCK, RAOUL ERNEST, M.B., 13, Buckingham Gate, S.W.

1879 Dennis, FREDERIC S., M.D., 542, Madison Avenue, New York, U.S.A.

1875 DENT, CLINTON T., 61, Brook Street, W. (C. 1884–6, V.P. 1905–7.) *Trans.* 5. *C.C.* 1.

O.M. DICKINSON, WILLIAM HOWSHIP, M.D., 10, Stanhope Place, Marble Arch, W. (C. 1874–5, V.P. 1888–9.) *Trans.* 2.

1894 Dickson, THOMAS HUGH, M.B., B.C., H.M. Customs, Lower Thames Street, E.C.

1871 DIVER, EBENEZER, M.D., 95, London Road, Portsmouth. (C. 1890–2. *Trans.* 2.

1901 DOUGLAS, JOHN J., M.D., 42, Central Hill, Norwood, S.E. *C.C.* 1.

1868 DRAGE, CHARLES, M.D., Hatfield, Herts.

1896 DREW, DOUGLAS, M.D., B.S., 1, Harley Street, Cavendish Square, W. *Trans.* 1. *C.C.* 4.

1906 DRYSDALE, J. H., M.D., 11, Devonshire Place, W.

O.M. DUCKWORTH, Sir DYCE, M.D., LL.D., 28, Grosvenor Place, S.W. (C. 1875–7, V.P. 1887–8, P. 1891–2.) *Trans.* 24, *C.C.* 8.

1902 DUDGEON, LEONARD S., 6, Powis Gardens, Bayswater, W. *Trans.* 1, *C.C.* 2.

1884 DUKE, EDGAR, 11, Wilbury Road, Hove.

1869 DUKE, OLLIVER THOMAS, M.B., Surgeon, Bengal Army, India.

1899 DUNCAN, ANDREW, M.D., 24, Chester Street, Grosvenor Place, S.W. *Trans.* 1.

1889 DUNCAN, JOHN, M.D., St. Petersburg.

1889 DUNN, LOUIS ALBERT, M.S., 51, Devonshire Street, Portland Place, W. (C. 1899–1902.) *Trans.* 2, *C.C.* 2.

1884 EDMUNDS, WALTER, M. C., 2, Devonshire Place, Portland Place, W. (C. 1902–5.) *Trans.* 2.

1899 EID, ALFRED, M.D., Cairo, Egypt.

1902 ELLIOT, ANDREW, M.D., 110, Gloucester Place, Portman Square, W.

ELECTED

1882 EMOND, EMILE, M.D., 113, Boulevard Beaumarchais, Paris.

1902 ENGLISH, THOMAS CRISP, B.S., 31, Grosvenor Street, W. *C.C.* 1.

1893 EVANS, WILLMOTT H., M.D., B.S., 2, Upper Wimpole Street, Cavendish Square, W. *C.C.* 1.

1888 **EVE**, FREDERIC S., 61, Harley Street, Cavendish Square, W. (C. 1899–1902.) *Trans.* 6, *C.C.* 10.

1877 **EWART**, WILLIAM, M.D., 33, Curzon Street, W. (C. 1884–6, V.P. 1905–7.) *Trans.* 5, *C.C.* 4.

1899 FAGGE, CHARLES HERBERT, M.S., 3, Devonshire Place, Portland Place, W. (C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 2, *C.C.* 1.

1868 **Fairbank**, FREDERICK ROYSTON, M.D., Westcott, Dorking, Surrey. *Trans.* 2.

1905 FAIRBANK, HAROLD A. T., 137, Harley Street, W. *C.C.* 1.

1900 FAWCETT, JOHN, M.D., 66, Wimpole Street, W. *Trans.* 3, *C.C.* 2.

1902 FEDDEN, WALTER FEDDE, M.B., M.S., 20, Lower Seymour Street, Portman Square, W.

1902 FENNELL, CHARLES HENRY, M.D., County Asylum, Hellingly, Sussex. *C.C.* 1.

1887 FENWICK, E. HURRY, 14, Savile Row, W. *Trans.* 2, *C.C.* 2.

1872 FENWICK, J. C. J., M.D., Long Framlington, Morpeth.

1893 FENWICK, WILLIAM SOLTAU, M.D., 29, Harley Street, W. *Trans.* 2.

1878 FIELD, GEORGE P., 152, Harley Street, W.

1876 FINLAY, Prof. DAVID WHITE, M.D., 2, Queen's Terrace, Aberdeen. (C. 1885–7, S. 1891.) *Trans.* 5.

1894 FLETCHER, HERBERT MORLEY, M.D., 98, Harley Street, W. (C. 1906–7. C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 2, *C.C.* 1.

1878 **FONMARTIN**, HENRY DE, M.D. (Travelling.)

1905 FORSYTH, DAVID, M.D., 19, Harley Street, W. *Trans.* 1.

1890 FOSTER, MICHAEL G., M.D., M.A., Villa San Giovanni, San Remo, Italy.

1899 FOWLER, CHARLES OWEN, M.D., Cotford House, Thornton Heath, Surrey.

1881 FOWLER, JAMES KINGSTON, M.D. (*Trustee* 1906–7), 35, Clarges Street, W. (C. 1887–9, 1899–1902, S. 1896–9, V.-P. 1904–6.) *Trans.* 4, *C.C.* 6.

1886 FOX, R. HINGSTON, M.D., 29, Weymouth Street, Portland Place, W. *C.C.* 1.

1878 FOX, THOMAS COLCOTT, M.B., 14, Harley Street, W. (C. 1888–90.) *Trans.* 7, *C.C.* 2.

1893 FOXWELL, ARTHUR, M.D., 22, Newhall Street, Birmingham.

1902 FRENCH, HERBERT, M.D., 26, St. Thomas's Street, London Bridge, S.E. *Trans.* 2. *C.C.* 1.

1897 FRIPP, Sir ALFRED DOWNING, C.B., M.V.O., M.S., 19, Portland Place, W. (C. 1904–7.) *Trans.* 3.

1899 FÜRTH, KARL, M.D., 39, Harley Street, W.

1890 FULLER, HENRY ROXBURGH, M.D., 45, Curzon Street, W.

ELECTED

1897 FURNIVALL, PERCY, 28, Weymouth Street, Portland Place, W.

1891 FYFFE, W. KINGTON, M.B., B.C., 19, Duke Street, Manchester Square, W. (Travelling, New Zealand.) *Trans.* 1.

1895 GALLOWAY, JAMES, M.D., 54, Harley Street, W.

1887 GARROD, ARCHIBALD EDWARD, M.A., M.D. (*Hon. Secretary*), 9, Chandos Street, Cavendish Square, W. (C. 1898–1901, S. 1905–7. Sec. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 5, *C.C.* 7.

1879 GARSTANG, THOMAS WALTER HARROPP, Edge Mount, Altrincham.

1903 GATES, EDWARD A., via Palestro A., Florence.

1885 GIBBONS, ROBERT ALEXANDER, M.D., 29, Cadogan Place, S.W. *Trans.* 1.

1893 GIBBS, CHARLES, 48, Queen Anne Street, W. *C.C.* 1.

1893 GLOVER, LEWIS G., M.B., B.C., 17, Belsize Park, Hampstead, N.W.

1875 GODLEE, RICKMAN JOHN, M.S., 19, Wimpole Street, W. (C. 1882–3, 1887–9, S. 1884–6, V.P. 1895–8.) *Trans.* 14, *C.C.* 11.

1901 GOFFE, ERNEST GEORGE LEOPOLD, M.D., North-Eastern Hospital, 81, St. Ann's Road, Tottenham, N.

1882 GOLDIE, ROBERT WILLIAM, Hirst House, Hirst, Morpeth, Northumberland.

1878 **GOLDING-BIRD**, C. H., M.B., 12, Queen Anne Street, W. (C. 1887–9, V.-P. 1903–5.) *Trans.* 18, *C.C.* 1.

1894 GOODALL, EDWARD WILBERFORCE, M.D., Eastern Hospital, Homerton, N.E. (C. 1904–7.) *Trans.* 8.

1875 GOODHART, JAMES FREDERIC, M.D., 25, Portland Place, W. (C. 1880–2, V.P. 1897–1900.) *Trans.* 14, *C.C.* 2.

1891 GOODMAN, ROGER NEVILLE, M.B., Elmside, Kingston-on-Thames.

1869 GOODRIDGE, HENRY FREDERICK AUGUSTUS, M.D., 10, Brock Street, Bath.

1877 GOULD, A. PEARCE, M.S., 10, Queen Anne Street, W. (C. 1885–7, 1895–8, S. 1892–5, V.-P. 1898–1901.) *Trans.* 14, *C.C.* 9.

1901 GOWAN, BOWIE CAMPBELL, Raven Dene, Great Stanmore, Middlesex.

1875 GOWERS, Sir WILLIAM RICHARD, M.D., F.R.S., 50, Queen Anne Street, W. (C. 1881–2, V.-P. 1903–5.) *Trans.* 5, *C.C.* 1.

1907 GRAHAM, CECIL IRVING, 49, Marloes Road, S.W.

1904 GRAHAM, GEORGE HERBERT, M.D., 14, Old Cavendish Street, W.

1891 GRANT, J. DUNDAS, M.D., 18, Cavendish Square, W.

1875 Greenfield, WILLIAM SMITH, M.D., 7, Heriot Row, Edinburgh. (C. 1881.) *Trans.* 3.

1893 **GRIFFITH**, WALTER SPENCER ANDERSON, M.D., 96, Harley Street, W.

1895 Grube, KARL, M.D., Neuenahr, Germany. *Trans.* 1.

1895 GUTHRIE, LEONARD GEORGE, M.D., 15, Upper Berkeley Street, W. (C. 1906–7. C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 2, *C.C.* 9.

1887 HABERSHON, SAMUEL HERBERT, M.D., 88, Harley Street, W. (C. 1901–4.) *C.C.* 1.

ELECTED

1900 HADLEY, WILFRED J., M.D., 33, Queen Anne Street, Cavendish Square, W.

1875 HALE, C. D. B., 3, Sussex Place, W. *Trans.* 1.

1903 HALL, JOHN BASIL, M.C., 116, Manningham Lane, Bradford.

1878 **HALL**, F. DE HAVILLAND, M.D., 47, Wimpole Street, W. (C. 1885–7, V.-P. 1904–6.) *Trans.* 9, *C.C.* 5.

1889 HALSTEAD, GEORGE EZRA, M.D., B.S., Albion Hill House, Ramsgate.

1888 HANDFIELD-JONES, MONTAGU, M.D., 35, Cavendish Square, W.

1886 **Handford**, HENRY, M.D., Elmfield, Southwell. (C. 1893–4.) *Trans.* 8, *C.C.* 1.

1906 HANN, REGINALD GEORGE, Sherwood House, Blundell Street, Leeds.

1904 HARBURN, JOHN ENGLISH, 8, Broad Walk, Buxton, Derbyshire.

1886 HARDIE, JAMES, M.D., 15, St. John Street, Manchester.

1890 HARPER, JAMES, M.D., 25, Rosary Gardens, South Kensington, S.W.

1872 HARRIS, HENRY, M.D., Trengweath, Redruth, Cornwall.

1889 HARRIS, HERBERT ELWIN, M.B., 13, Lansdown Place, Victoria Square, Clifton, Bristol. *Trans.* 1.

1903 HARRIS, WILFRED, M.D., 61, Wimpole Street, Cavendish Square, W. *Trans.* 1, *C.C.* 2.

1881 HARRISON, CHARLES EDWARD, M.B., 19, Westgate Terrace, Redcliffe Square, S.W.

1892 HARRISSON, DAMER, 53, Rodney Street, Liverpool. *Trans.* 1.

1901 HARTLEY, PERCIVAL HORTON-SMITH, M.V.O., M.D., 19, Devonshire Street, Portland Place, W.

1869 **HAWARD**, J. WARRINGTON, 57, Green Street, Grosvenor Square, W. (C. 1876–8, 1884–6, S. 1881–3, V.P. 1888.) *Trans.* 15.

1899 **Hawkes**, CLAUDE SOMERVILLE, Glencairn, Wickham Terrace, Brisbane, Queensland, Australia.

1886 HAWKINS, FRANCIS HENRY, M.D., 73, London Street, Reading. (C. 1902–5.) *Trans.* 8, *C.C.* 3.

1889 HAWKINS, HERBERT PENNELL, M.D., B.Ch., 56, Portland Place, W. (C. 1900–3.) *Trans.* 1.

1890 HAWKINS-AMBLER, GEORGE ARTHUR, 30, Rodney Street, Liverpool.

1900 HAYWARD, JOHN ARTHUR, M.D., 23, The Grange, Wimbledon Common, S.W.

1903 HEATH, PHILIP MAYNARD, M.B., 36, Cavendish Square, W. *C.C.* 1.

1879 HENDERSON, GEORGE COURTENAY, M.D., Kingston, Jamaica, West Indies.

1884 HERRINGHAM, WILMOT PARKER, M.D., 40, Wimpole Street, W. (C. 1894–7.) *Trans.* 6, *C.C.* 4.

1874 HOLDERNESS, WILLIAM BROWN, 15, Park Street, Windsor.

1868 **Holman**, Sir CONSTANTINE, M.D., 26, Gloucester Place, Portman Square, W. (C. 1894–7.)

1878 HOOD, DONALD WILLIAM CHARLES, M.D., 43, Green Street, Park Lane, W. (C. 1891–3.) *Trans.* 1.

ELECTED

1884 HORSLEY, Sir VICTOR, M.B., F.R.S., 25, Cavendish Square, W. (C. 1894.) *Trans.* 2, *C.C.* 1.

1895 HOUGH, CHARLES HENRY, The Cottage, Clappersgate, Ambleside, Windermere.

1880 **HOVELL**, T. MARK, 105, Harley Street, W. *Trans.* 1.

1893 HOWARD, R. J. BLISS, M.D., 31, Queen Anne Street, W.

1898 HULKE, SYDNEY BACKHOUSE, 162, Holland Road, Kensington, W.

1897 HUMPHRYS, CHARLES B., Eagle House, Blandford, Dorset.

1897 HUNT, GEORGE BERTRAM, M.B., 47, Albemarle Crescent, Scarborough, *Trans.* 1, *C.C.* 1.

1892 HUNTER, WILLIAM, M.D., 103, Harley Street, W.

O.M. HUTCHINSON, JONATHAN, F.R.S., 15, Cavendish Square, W. (C. 1867–8. V.P. 1875–6.) *Trans.* 14, *C.C.* 1.

1896 HUTCHINSON, JONATHAN, jun., 15, Cavendish Square, W. (C. 1902–5.) *Trans.* 6, *C.C.* 8.

1893 IONIDES, THEODORE HENRY, M.B., B.S., 25, First Avenue, Brighton.

1883 JACKSON, GEORGE HENRY, Ashburton, Carew Road, Eastbourne.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.S., 3, Manchester Square, W. (C. 1872–3, V.P. 1887.) *Trans.* 2, *C.C.* 1.

1888 JAMES, JAMES THOMAS, M.D., 108, Harley Street, W.

1901 JOHNSON, EDWARD ANGAS, M.B., B.S., 56, Franklin Street, Adelaide, South Australia.

1907 JOHNSON, HENRY PERCIVAL, Military Hospital, Devonport.

1889 JOHNSON, RAYMOND, M.B., B.S. (*Hon. Secretary*), 11, Wimpole Street, W. (C. 1900–3. S. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 7, *C.C.* 6.

1893 JOHNSTON, G. F., M.D., 3, Montagu Place, Montagu Square, W.

1904 JONES, A. ERNEST, M.D., B.Sc., 13, Harley Street, W.

1903 JONES, ERNEST LLOYD, 59, Trumpington Street, Cambridge.

1902 JONES, LAWRENCE, 47, Upper Brook Street, W.

1898 Jones, ROBERT, 11, Nelson Street, Liverpool. *Trans.* 2.

1872 JONES, THOMAS RIDGE, M.D., 4, Chesham Place, S.W. (C. 1892–3.)

1876 JORDAN, FURNEAUX, 10, Ferndale, Teignmouth, S. Devon. *Trans.* 1.

1902 JOWERS, REGINALD F., 55, Brunswick Square, Brighton.

1878 KEETLEY, CHARLES ROBERT BELL, 56, Grosvenor Street, W. (C. 1895–8.) *Trans.* 2.

1898 KELLOCK, THOMAS HERBERT, 8, Queen Anne Street, Cavendish Square, W. (C. 1903–6.) *Trans.* 5, *C.C.* 9.

1901 KELYNACK, THEO. N., M.D., 120, Harley Street, W.

1897 KEYSER, CHARLES RALPH, Butler's Green, Haywards Heath, Sussex. *Trans.* 1, *C.C.* 5.

1883 KIDD, PERCY, M.D., 60, Brook Street, W. (C. 1891–2, 1902–5, S. 1899–1902.) *Trans.* 5, *C.C.* 10.

1887 Knaggs, R. LAWFORD, B.C., 27, Park Square, Leeds. *Trans.* 2.

1878 LACEY, THOMAS WARNER, 196, Burrage Road, Plumstead.

ELECTED

1897 LAMPLOUGH, CHARLES, Kirkstall, Alverstoke, Hants.

1890 LANCASTER, ERNEST LE CRONIER, M.B., B.Ch., Winchester House, Swansea, S. Wales. *Trans.* 2.

1895 LANE, JAMES ERNEST, 47, Queen Anne Street, W. (C. 1905–7. C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 1.

1883 LANE, WILLIAM ARBUTHNOT, M.B., M.S., 21, Cavendish Square, W. (C. 1893–6.) *Trans.* 29, *C.C.* 10.

O.M. **LANGTON**, JOHN, 62, Harley Street, W. (C. 1878–80, V.P. 1892–4, P. 1897–9.) *Trans.* 5.

1886 LANKESTER, HERBERT, M.D., Ingleborough, Woking, Surrey.

1907 LAWRENCE, H. GWYNNE, M.B., 49, Hill Street, W.

1893 LAWSON, ARNOLD, M.D., 12, Harley Street, W.

1900 LEAF, CECIL H., M.B., 75, Wimpole Street, Cavendish Square, W.

1877 **Lediard**, HENRY AMBROSE, M.D., 26, Lowther Street, Carlisle. (C. 1889, V.-P. 1902–4.) *Trans.* 5, *C.C.* 1.

1896 **Leech**, PRIESTLEY, M.D., King Cross, Halifax.

1877 LEES, DAVID B., M.D., 22, Weymouth Street, W. (C. 1885, V.-P. 1902–4.) *Trans.* 6.

1902 LEGG, THOMAS PERCY, M.B., 141, Harley Street, Cavendish Square, W.

1899 LESLIE, R. MURRAY, M.D., 26, Harley Street, Cavendish Square, W. *C.C.* 1.

1901 LEWERS, ARTHUR HAMILTON NICHOLSON, M.D., 43, Upper Brook Street, W.

1892 LEWIS, EDWARD JOHN, M.B., B.C., 74, Hamilton Terrace, N.W.

1895 LEWIS, ERNEST E., M.D., 30, Weymouth Street, W.

1903 LITTLE, E. GRAHAM, M.D., 61, Wimpole Street, W.

1890 LITTLE, JOHN FLETCHER, M.B., 125, Harley Street, W. *C.C.* 5.

1891 LITTLEWOOD, HARRY, 25, Park Square, Leeds. *Trans.* 1.

1885 LOCKWOOD, CHARLES BARRETT, 19, Upper Berkeley Street, W. (C. 1896-9.) *Trans.* 7.

1876 **LONGHURST**, ARTHUR EDWIN TEMPLE, M.D., 26, Lower Belgrave Street, Eaton Square, S.W. (C. 1889–91.) *Trans.* 1.

1904 LONGHURST, FREDERIC W., 26, Lower Belgrave Street, Eaton Square, S.W.

1897 LOW, HAROLD, M.B., 10, Evelyn Gardens, South Kensington, S.W.

1899 LOW, V. WARREN, M.D., B.S., 146, Harley Street, Cavendish Square, W. (C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 1.

1881 LUBBOCK, MONTAGU, M.D., 19, Grosvenor Street, W.

1876 LUCAS, R. CLEMENT, M.B., B.S., 50, Wimpole Street, W. (C. 1883–5, V.P. 1901–3.) *Trans.* 12. *C.C.* 1.

1894 LUFF, ARTHUR PEARSON, M.D., 9, Queen Anne Street, Cavendish Square, W.

1879 LUNN, JOHN REUBEN, St. Marylebone Infirmary, Rackham Street, Ladbroke Grove Road, W. (C. 1890–1, V.P. 1905–7.) *Trans.* 9, *C.C.* 25.

ELECTED

1893 LYE, HENRY GRABHAM, M.D., Southbrook, Suffolk Road, Bournemouth.
1889 MACBRIDE, P., M.D., 16, Chester Street, Edinburgh.
1891 MACDONALD, GREVILLE, M.D., 85, Harley Street, W.
1901 MCGAVIN, LAURIE HUGH, 6, Mansfield Street, Cavendish Square, W. *Trans.* 5, *C.C.* 6.
1881 MCHARDY, MALCOLM MACDONALD, 5, Savile Row, W. *Trans.* 1.
1891 MACKENZIE, HECTOR W. G., M.A., M.D., 34, Upper Brook Street, W. (C. 1902–5.) *Trans.* 6, *C.C.* 1.
1879 MACKENZIE, SIR STEPHEN, M.D., 18, Cavendish Square, and Merrycourt, Gt. Bookham, Leatherhead. (C. 1884, 1888–90, S. 1885–7. V.P. 1900–2.) *Trans.* 9, *C.C.* 10.
1884 MACKERN, JOHN, M.B., St. German's Lodge, Shooter's Hill Road, Blackheath, S.E.
1885 MACLAREN, RODERICK, M.D., Portland Square, Carlisle. *Trans.* 1.
1879 MAGILL, JAMES, M.D., M.C., Coldstream Guards, Queen Anne's Mansions, S.W.
1885 MAGUIRE, ROBERT, M.D., 4, Seymour Street, W. *Trans.* 1.
1881 MAKINS, GEORGE HENRY, C.B., 47, Charles Street, Berkeley Square, W. (C. 1892–4, 1898–1901, S. 1895–8, T. 1903–7. V.P. 1906–7. V.-P. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 6, *C.C.* 7.
1887 MALCOLM, JOHN D., M.B., C.M., 13, Portman Street, W. (C. 1900–3.) *Trans.* 3, *C.C.* 2.
1890 MANSON, Sir PATRICK, K.C.M.G., M.D., C.M., F.R.S., 21, Queen Anne Street, W. (C. 1895–7.) *C.C.* 1.
1902 MARRIOTT, CECIL, M.B., M.C., 11, Welford Road, Leicester.
1888 Marriott, HYDE, M.B., Dial House, Stockport.
1868 MARSH, Professor F. HOWARD, 14, Hertford Street, Mayfair, W. (C. 1876–7, 1881–3, S. 1878–80, V.P. 1887–8, T. 1899–1901, P. 1901–3.) *Trans.* 20, *C.C.* 7.
1887 MARTIN, SIDNEY, M.D., B.S., F.R.S., 10, Mansfield Street, W. (C. 1896–9.)
1888 MASON, DAVID JAMES, M.D., C.M., Rosemont, Maidenhead.
1892 MASTERS, JOHN ALFRED, M.D., 94, Knightsbridge, S.W.
1884 MAUDSLEY, HENRY CARR, M.D., 22, Collins Street, Melbourne, Victoria.
1897 MAY, CHICHESTER GOULD, M.D., 59, Cadogan Place, S.W.
1868 May, EDWARD HOOPER, M.D., High Cross, Tottenham, Middlesex.
1888 MAY, WILLIAM PAGE, M.D., B.Sc., 9, Manchester Square, W.; and Helouan, nr. Cairo, Egypt (October to April).
1906 MAYNARD, JOHN SEYMOUR, M.D., 4, Charles Street, St. James's Square, S.W.
1901 MENZIES, F. U., Edinburgh University Union, Edinburgh.
1888 MENZIES, J. HERBERT, 10, Wetherby Place, S.W.
1893 MERCER, WILLIAM BRACEWELL, M.B., B.C., Cliffe Cottage, Ripponden, near Halifax, Yorks.
1878 MEREDITH, WILLIAM APPLETON, C.M., 21, Manchester Square, W. (C. 1887–9.) *Trans.* 2.

ELECTED

1894 MICHELS, ERNST, M.D., 48, Finsbury Square, E.C.

1890 MILEY, MILES, M.A., M.B., 21, Belsize Avenue, Hampstead, N.W.

1896 MILLS-ROBERTS, ROBERT HERBERT, C.M.G., Llanberis, North Wales. *Trans.* 1.

1902 MOON, R. O., M.D., 45, Green Street, Grosvenor Square, W. *C.C.* 1.

1874 **MORGAN**, JOHN HAMMOND, C.V.O. (*Trustee* 1902–7), 68, Grosvenor Street, W. (C. 1883–5, V.P. 1898–1901.) *Trans.* 2, *C.C.* 8.

1888 MORISON, ALEXANDER, M.D., 14, Upper Berkeley Street, W. *Trans.* 2, *C.C.* 1.

1899 MORISON, RUTHERFORD, 14, Saville Row, Newcastle-on-Tyne. *Trans.* 1.

1903 MORLEY, ARTHUR S., 29, Gower Street, W.C.

1877 MORRIS, HENRY, M.A., M.B., 8, Cavendish Square, W. (C. 1884–6. V.P. 1903–5.) *Trans.* 9.

1877 MORRIS, MALCOLM ALEX., 8, Harley Street, W. (C. 1890–2.) *Trans.* 1, *C.C.* 1.

1901 MORSE, THOMAS HENRY, All Saints' Green, Norwich. *Trans.* 1.

1902 MORTON, CHARLES ALFRED, 14, Vyvyan Terrace, Clifton, Bristol. *Trans.* 1.

1899 MOUAT-BIGGS, CHARLES E. FORBES, 2, Fauconberg Villas, Bayshill, Cheltenham. *C.C.* 2.

1879 MOULLIN, CHARLES W. MANSELL, 69, Wimpole Street, W. (C. 1888–90.) *Trans.* 11, *C.C.* 4.

1900 MOYNIHAN, BERKELEY GEORGE ANDREW, M.S., 33, Park Square, Leeds. (C. 1904–7.) *Trans.* 4.

1905 MUECKE, FRANCIS FREDERICK, M.B., B.S., 63, Wellington Road, N.W.

1875 MURPHY, Sir SHIRLEY F., 9, Bentinck Terrace, Regent's Park, N.W. (C. 1888–90.) *C.C.* 1.

1885 MURRAY, ALEXANDER DALTON, M.B., Colombo, Ceylon.

1893 MURRAY, GEORGE REDMAYNE, M.D., 11, Ellison Place, Newcastle-on-Tyne.

1883 MURRAY, HUBERT MONTAGUE, M.D., 25, Manchester Square, W. (C. 1893–6.) *Trans.* 1.

1894 MURRAY, JOHN, 110, Harley Street, W. *C.C.* 1.

1872 MYRTLE, ANDREW S., M.D., 10, Park Parade, Harrogate. (C. 1892.)

1902 NABARRO, DAVID, M.D., 4, Albemarle Mansions, Heath Drive, Hampstead, N.W.

1892 NASH, WALTER GIFFORD, 31, St. Peter's, Bedford.

1889 Newman, D., M.D., 28, Woodside Place, Glasgow. *Trans.* 5.

O.M. NORTON, ARTHUR TREHERN, C.B., Leyfields Wood, Ashampstead, Berks. (C. 1874–6.) *Trans.* 7.

1880 O'CONNOR, BERNARD, M.D., Twyford Abbey, Willesden, N.W. *Trans.* 1.

1899 OGLE, CYRIL, M.D., 96, Gloucester Place, Portman Square, W. *Trans.* 1.

1887 Oliver, THOMAS, M.D., 7, Ellison Place, Newcastle-upon-Tyne.

ELECTED

1887 OPENSHAW, THOMAS HORROCKS, C.M.G., M.B., 16, Wimpole Street, W. (C. 1905–7. C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 1, *C.C.* 8.

1868 OPPERT, FRANZ, M.D., 128, Leipzigerstrasse, Friedenau, Germany. *Trans.* 1.

1889 ORD, W. WALLIS, M.D., B.Ch., The Hall, Salisbury. *Trans.* 1, *C.C.* 1.

1887 ORMEROD, JOSEPH ARDERNE, M.D., 25, Upper Wimpole Street, W. (C. 1899–1902.) *C.C.* 2.

1884 ORMSBY, Sir LAMBERT HEPENSTAL, M.D., 92, Merrion Square West, Dublin.

1888 PAGE, FREDERICK, M.D., 1, Saville Place, Newcastle-on-Tyne.

1875 PAGE, HERBERT W., M.C., M.B., 146, Harley Street, W. (C. 1882–4, V.P. 1899–1902.) *Trans.* 8, *C.C.* 2.

1884 PAGET, STEPHEN, 70, Harley Street, W. (C. 1898–1901.) *Trans.* 9, *C.C.* 3.

1881 PARKER, RUSHTON, M.B., B.S., 59, Rodney Street, Liverpool. (C. 1896–9.) *Trans.* 1, *C.C.* 2.

1890 PARKIN, ALFRED, M.S., 24, Albion Street, Hull. *Trans.* 2.

1894 PARKINSON, JOHN PORTER, M.D., 57, Wimpole street, W. *Trans.* 1, *C.C.* 5.

1898 PARTRIDGE, ARTHUR A. H., M.D., M.Ch., St. Germans, Sutton, Surrey.

1881 PASTEUR, WILLIAM, M.D., 4, Chandos Street, W. (C. 1891–3, 1905–6, S. 1902–5, V.P. 1906–7. V.-P. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 7, *C.C.* 5.

1893 PATERSON, DONALD ROSE, M.D., C.M., 15, St. Andrew's Crescent, Cardiff.

1900 PATON, EDWARD PERCY, M.S., 53, Queen Anne Street, Cavendish Square, W. *Trans.* 3.

1892 PAUL, FRANK THOMAS, 38, Rodney Street, Liverpool. *Trans.* 1.

1893 PAUL, JOHN ERNEST, M.B., c/o Messrs. Parry & Co., 70, Gracechurch Street, E.C. *Trans.* 1.

O.M. PAVY, FREDERICK WILLIAM, M.D., F.R.S., 35, Grosvenor Street, W. (C. 1869–71, V.P. 1882–4.) *Trans.* 3.

1879 PEEL, ROBERT, 120, Collins Street East, Melbourne, Victoria.

1882 PEPPER, AUGUSTUS JOSEPH, M.S., M.B., 13, Wimpole Street, W. *Trans.* 1.

1898 PERKINS, J. J., M.D., 8, Mansfield Street, W. *Trans.* 1, *C.C.* 1.

1895 PHEAR, ARTHUR G., M.D., B.C., 47, Weymouth Street, Portland Place, W. *Trans.* 2.

1895 PHILLIPS, F. B. WILLMER, M.B., 7, Harper Place, Bedford.

1885 PHILLIPS, SIDNEY, M.D., 3, Upper Brook Street, W. (C. 1893–6.) *Trans.* 10, *C.C.* 6.

1885 PITT, GEORGE NEWTON, M.D., 15, Portland Place, W. (C. 1894–6.) *Trans.* 7, *C.C.* 6.

1871 Playne, ALFRED, M.B., Maidenhead.

ELECTED

1884 POLLARD, BILTON, B.S., 24, Harley Street, W. (C. 1895–8.) *Trans.* 2

1868 POLLOCK, JAMES EDWARD, M.D., 37, Collingham Place, S.W. (C. 1878–80.)

1881 POWELL, H. A., M.A., c/o Messrs. Powell and Burt, 28 and 29, St. Swithin's Lane, E.C.

O.M. POWELL, Sir R. DOUGLAS, Bart., K.C.V.O., M.D., 62, Wimpole Street, W. (C. 1874–6, V.P. 1889–90, P. 1899-1901.) *Trans.* 4.

1868 PRENTIS, CHARLES, Surgeon-Major, Bengal Medical Service; India.

1902 PRICE, F. W., M.B., 133, Harley Street, W. *Trans.* 1.

1884 PRINGLE, JOHN JAMES, M.B., 23, Lower Seymour Street, W. (C. 1890-2, 1897–1900.) *Trans.* 1, *C.C.* 3.

1884 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., 48, Brook Street, W. (C. 1890–2.) *Trans.* 1.

1896 PYE-SMITH, RUTHERFORD JOHN, 350, Glossop Road, Sheffield.

1893 RAKE, ALFRED THEODORE, M.B., B.S., 58, Delancey Street, Regent's Park, N.W.

1895 RAMSAY, HERBERT MURRAY, 35A, Hertford Street, Mayfair, W.

1889 RANKING, JOHN E., M.D., Hanover House, Tunbridge Wells.

1883 READ, THOMAS LAURENCE, 11, Petersham Terrace, Queen's Gate, S.W.

1902 REID, Sir JAMES, Bart., G.C.V.O., K.C.B., 72, Grosvenor Street, Grosvenor Square, W.

1901 REISSMANN, CHARLES, M.D., 8, North Terrace, Adelaide, South Australia.

1899 Rennie, GEORGE EDWARD, M.D., College Street, Hyde Park, Sydney, N.S.W.

1868 RICE, MICHAEL W., M.D., Elmbank, Hargrave, Northamptonshire, (C. 1876–8.)

1898 RICHARDS, JOSEPH STEWART, M.D., B.S., 5, Wellesley Villas, Wellesley Road, Croydon, S.E. *C.C.* 2.

1903 RIGBY, HUGH, M.S., 7, Wimpole Street, Cavendish Square, W. *C.C.* 2.

O.M. RINGER, SYDNEY, M.D., F.R.S., 15, Cavendish Place. W. (C. 1871–2.) *Trans.* 1.

1896 RISDON, WILLIAM ELLIOT, M.D., 4, Etchingham Park Road, Church End, Finchley, N.

1873 Roberts, DAVID LLOYD, M.D., 11, St. John Street, Manchester.

1888 ROBERTS, FRANK ERNEST, Tulse Dale Villa, Lower Norwood, S.E.

1883 ROBERTS, FREDERICK THOMAS, M.D., 102, Harley Street, W. (C. 1892–4.)

1900 ROBERTS, GRIFFITH LLOYD, M.B., C.M., Castle Square, Carnarvon, North Wales.

1890 ROBERTSON, ROBERT, M.D., Belgrave Road, Ventnor, Isle of Wight.

1885 ROBINSON, ARTHUR HENRY, M.D., St. Mary's Infirmary, Highgate Hill, N. *C.C.* 3.

1892 ROBINSON, HENRY BETHAM, M.D., M.S., 1, Upper Wimpole Street, W. (C. 1901–4). *Trans.* 11, *C.C.* 12.

ELECTED

1885 ROBSON, A. W. MAYO, 8, Park Crescent, W., (C. 1893–6.) *Trans.* 15.

1889 ROLLESTON, HUMPHRY DAVY, M.A., M.D., 55, Upper Brook Street, Grosvenor Square, W. (C. 1899–1902.) *Trans.* 11. *C.C.* 10.

1897 ROSS, FREDERICK WILLIAM FORBES, M.D., 15, Gower Street, W.C. *C.C.* 1.

1877 ROTH, BERNARD, 38, Harley Street, Cavendish Square, and "Kingswood," Enfield, Middlesex. *Trans.* 1, *C.C.* 4.

1890 ROUGHTON, EDMUND WILKINSON, B.S., 38, Queen Anne Street, W. *Trans.* 2, *C.C.* 1.

1905 ROUGHTON, JOHN PAUL, Evelyns, Kettering, Northamptonshire. *Trans.* 1.

1874 ROWLAND, EDWARD ROGER, Dordrecht, Wodehouse, S. Africa.

1904 ROWLANDS, R. P., M.S., 6, St. Thomas's Street, London Bridge, S.E. *C.C.* 2.

1906 ROWNTREE, CECIL W., B.S., 16, Weymouth Street, W. *Trans.* 1.

1898 RUSSELL, ALBRED ERNEST, M.D., B.S., 9, Wimpole Street, W. *C.C.* 1.

1900 RUSSELL, J. S. RISIEN, M.D., 44, Wimpole Street, Cavendish Square, W. *Trans.* 1, *C.C.* 1.

1903 RUSSELL, JAMES WILLIAM, M.D., 72, Newhall Street, Birmingham.

1887 RUTHERFOORD, H. T., M.B., Salisbury House, Taunton.

1907 RYALL, CHARLES, 62, Harley Street, W.

1885 RYLE, REGINALD JOHN, M.D., 15, German Place, Brighton.

1899 SANDIFER, HENRY STEPHEN, M.D., B.S., 11, Porchester Gardens, W.

1873 SAVAGE, GEORGE HENRY, M.D., 26, Devonshire Place, W. (C. 1882–3.)

1886 SAVILL, THOMAS DIXON, M.D., 60, Upper Berkeley Street, W. *Trans.* 1, *C.C.* 6.

1886 SCOTT, ALFRED, 15, German Place, Brighton.

1894 SCOTT, BERNARD, "Hartington," Poole Road, Bournemouth.

1892 SCOTT, RICHARD JAMES HERBERT, 28, Circus, Bath. *C.C.* 1.

1902 SEARS, ALFRED ERNEST, 33, Lee Terrace, Blackheath, S.E. *Trans.* 1.

1897 SEGUNDO, CHARLES SEMPILL DE, M.B., B.S., 97, Gloucester Place, Portman Square, W.

1892 SELWYN-HARVEY, JOHN STEPHENSON, M.D., 1, Astwood Road, S.W.

1878 SEMON, Sir FELIX, C.V.O., M.D., 39, Wimpole Street, W. (C. 1885–7. V.-P. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 8, *C.C.* 3.

1884 SHARKEY, SEYMOUR J., M.D., 22, Harley Street, W. (C. 1895–8.)

1898 SHAW, HAROLD BATTY, M.D., 7, Devonshire Street, Portland Place, W. *C.C.* 10.

1889 SHAW, LAURISTON ELGIE, M.D., 64, Harley Street, W. *C.C.* 1.

1906 SHEEHEN, JAMES, 40, Devonshire Street, Portland Place, W. *Trans.* 4.

1875 SHERWOOD, ARTHUR PAUL, 8, Seaside Road, Eastbourne.

1903 SIMPSON, GRAHAM, The Sheffield Royal Hospital, Sheffield. *Trans.* 1 *C.C.* 1.

1872 SLIGHT, GEORGE, M.D., 14, Old Burlington Street, W.

ELECTED

1896 SMITH, EBENEZER STANLEY, M.D., 68, Wimpole Street, Cavendish Square, W.

1888 SMITH, FREDERICK J., M.D., 138, Harley Street, W. (C. 1905–7. C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 2.

1884 SMITH, R. PERCY, M.D., 36, Queen Anne Street, Cavendish Square, W. (C. 1898–1901.)

1894 SMITH, THOMAS RUDOLPH, M.B., B.C., 25, Bridge Road, Stockton-on-Tees.

1893 SNAPE, ERNEST ALFRED, M.D., 41, Welbeck Street, W.

1868 SNOW, WILLIAM V., M.D., Richmond Gardens, Bournemouth.

1890 SOLLY, ERNEST, M.B., Strathlea, Harrogate, Yorks. *C.C.* 1.

1888 SPENCER, WALTER GEORGE, M.S., M.B., 35, Brook Street, W. (C. 1896–9. C. Clin. Sect. Roy. Soc. Med., 1907– . S. 1904–7.) *Trans.* 16, *C.C.* 18.

1885 SPICER, FREDERICK, M.D., 17, Wimpole Street, Cavendish Square, W.

1888 SPICER, ROBERT HENRY SCANES, M.D., 28, Welbeck Street, W. *C.C.* 1.

1882 SPOONER, FREDERICK HENRY, M.D., Shameen, 33, Pembury Road, Lower Clapton, N.E.

1896 SPURRELL, CHARLES, Medical Superintendent, Poplar and Stepney Sick Asylum, Devon's Road, Bromley-by-Bow, E.

1876 SQUIRE, A. BALMANNO, 24, Weymouth Street, W. *Trans.* 5, *C.C.* 4.

1879 STAPLES, FRANCIS PATRICK, Brigade-Surgeon, Army (retired). (Address uncommunicated.)

1896 STEWARD, FRANCIS JAMES, M.B., B.S., 125, Harley Street, W. (C. 1906–7. C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 3, *C.C.* 1.

1874 Stirling, EDWARD C., M.D. [care of Messrs. ELDER & Co., 7, St. Helen's Place, E.C.], Adelaide, South Australia.

1899 STONE, WILLIAM GREAM, M.D., 93, Denmark Hill, S.E. *C.C.* 2.

1884 STONHAM, CHARLES, C.M.G., 4, Harley Street, W. *C.C.* 3.

1878 STRUGNELL, FREDERICK WILLIAM, Grove End House, Highgate Road, Highgate, N.W. (C. 1903–6.) *C.C.* 1.

1878 Sturge, WILLIAM ALLEN, M.D., Icklingham Hall, Mildenhall, Suffolk.

1894 SUTHERLAND, GEORGE A., M.D., 73, Wimpole Street, Cavendish Square, W. (C. 1906–7. C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 2, *C.C.* 3.

1882 SYMONDS, CHARTERS JAMES, M.S. (*Trustee* 1903–7), 58, Portland Place, W. (C. 1888–9, 1901–4, S. 1898–1901, V.P. 1906–7. V.-P. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 13, *C.C.* 24.

1876 SYMONDS, HORATIO PERCY, 35, Beaumont Street, Oxford.

1885 TAIT, EDWARD SABINE, M.D., 48, Highbury Park, N.

1885 TAIT, HENRY BREWER, Lincluden, Sunnyside Road, Hornsey Lane, N.

1891 TATE, WALTER WILLIAM HUNT, M.D., 32, Queen Anne Street, W.

1886 TAY, WAREN, 4, Finsbury Square, E.C.

ELECTED

1897 TAYLOR, E. CLAUDE, M.D., B.S., 181, Rosslyn Hill, Hampstead, N.W.

1875 **TAYLOR**, FREDERICK, M.D., 20, Wimpole Street, W. (S. 1879–81, C. 1882–4, V.P. 1894–7, T. 1901–3, P. 1903–5.) *Trans.* 17, *C.C.* 3.

1889 **Taylor**, HENRY HERBERT, 36, Brunswick Square, Hove, Sussex.

1890 TAYLOR, JAMES, M.D., 49, Welbeck Street, W. *C.C.* 3.

1882 TAYLOR, SEYMOUR, M.D., 16, Seymour Street, W. *Trans.* 1, *C.C.* 5.

1885 Taylor, W. C. EVERLEY, 34, Queen Street, Scarborough.

1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., North Grange, Headingley, Leeds. (C. 1897–1900.)

1896 TEMPLETON, GEORGE, M.B., 10, Seymour Street, Portman Square, W.

1890 THANE, EDGAR HERBERT, M.D., Wagga-Wagga, New South Wales.

1886 THOMPSON, CHARLES HERBERT, M.D., 133, Harley Street, Cavendish Square, W. *C.C.* 1.

1897 THOMSON, H. CAMPBELL, M.D., 34, Queen Anne Street, Cavendish Square, W. (C. Clin. Sect. Roy. Soc. Med., 1907– .) *C.C.* 1.

1894 THOMSON, STCLAIR, M.D., 28, Queen Anne Street, W. *Trans.* 2, *C.C.* 9.

1896 THORNE, WILLIAM BEZLY, M.D., 2, Harley Street, W. *C.C.* 2.

1872 THORNTON, WILLIAM PUGIN, 35, St. George's Road, Canterbury. *Trans.* 5.

1885 THURSFIELD, THOMAS WILLIAM, M.D., Selwood, Beauchamp Square, Leamington.

1899 TILLEY, HERBERT, M.D., 89, Harley Street, Cavendish Square, W. *C.C.* 2.

1891 TOMSON, W. BOLTON, M.D., Park Street West, Luton, Bedfordshire.

1892 TOOTH, HOWARD HENRY, C.M.G., M.D., 34, Harley Street, W. (C. 1905–7. C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 1.

1887 TOTSUKA, KANKAI, Tokio, Japan.

1897 TUBBY, ALFRED HERBERT, M.S., 68, Harley Street, W. (C. 1905–7. C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 2, *C.C.* 8.

1900 TULLIS, GEORGE AUGUSTUS, M.D., Denstone, Christ Church Road, Winchester.

1904 TURNER, GEORGE GREY, 22, Jesmond Road, Newcastle-on-Tyne.

1882 TURNER, GEORGE ROBERTSON, 41, Half Moon Street, W. (C. 1897–1900.) *Trans.* 9. *C.C.* 6.

1904 TURNER, PHILIP, M.S., 8, St. Thomas's Street, London Bridge, S.E.

1888 TURNER, PHILIP DYMOCK, M.D., Sudbury, Ryde, Isle of Wight. *Trans.* 2.

1898 TURNER, WILLIAM, M.S., 17, Harley Street, Cavendish Square, W. *C.C.* 1.

1893 TURNEY, HORACE GEORGE, M.D., 68, Portland Place, W. (C. 1906–7. C. Clin. Sect. Roy. Soc. Med., 1907– .) *C.C.* 6.

1878 TYSON, WILLIAM JOSEPH, M.D., 10, Langhorne Gardens, Folkestone. (C. 1886–8, V.-P. 1900–2.) *Trans.* 8.

1881 UHTHOFF, JOHN CALDWELL, M.D., Wavertree House, Brunswick Place, Brighton. (C. 1898–1901.)

ELECTED

1868 VENNING, Sir EDGCOMBE, 30, Cadogan Place, S.W. (C. 1876–8.) *Trans.* 2.

1890 VOELCKER, ARTHUR FRANCIS, M.D., B.S., 101, Harley Street, W. (C. 1900–3.) *Trans.* 3, *C.C.* 4.

1868 WAGSTAFFE, WILLIAM WARWICK, Cotswold, Dartford Road, Sevenoaks. (C. 1878.)

1886 **WAINEWRIGHT**, BENJAMIN, M.B., C.M. (address uncommunicated).

1885 WAKLEY, THOMAS, jun., 16, Hyde Park Gate, S.W.

1899 WALKER, HENRY ROE, 8, Harley Street, W.

1896 WALLACE, CUTHBERT SYDNEY, M.B., 26, Upper Wimpole Street, W. (C. 1902–5.) *Trans.* 8, *C.C.* 5.

1890 WALLIS, FREDERICK CHARLES, M.B., B.C., 107, Harley Street, W. (C. 1902–5.) *Trans.* 6, *C.C.* 10.

1888 WALTERS, FREDERICK RUFENACHT, M.D., Crooksbury Sanatorium, Farnham, Surrey. *C.C.* 2.

1896 WARD, EDWARD, M.B., 30, Park Square, Leeds. *Trans.* 1.

1904 WARING, HOLBURT J., M.S., 37, Wimpole Street, Cavendish Square, W.

1888 Warner, PERCY, Rydal, Woodford Green, Essex.

1891 WATERHOUSE, HERBERT FURNIVALL, M.D., C.M., 81, Wimpole Street, W. (C. 1905–7. C. Clin. Sect. Roy. Soc. Med., 1907– .)

1902 WATSON, CHARLES GORDON, 44, Welbeck Street, Cavendish Square, W. *C.C.* 3.

1896 **WEBER**, FRED. PARKES, M.D., 19, Harley Street, W. (C. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 3, *C.C.* 14.

O.M. WEBER, Sir HERMANN D., M.D., 10, Grosvenor Street, W. (C. 1867–71, V.P. 1873–5.) *Trans.* 9.

1882 WEST, SAMUEL, M.D., 15, Wimpole Street, W. (C. 1887–9.) *Trans.* 17, *C.C.* 5.

1895 WETHERED, FRANK J., M.D., 83, Harley Street, W.

1874 WHEELHOUSE, CLAUDIUS GALEN, Cliffe Point, Filey, Yorks. *Trans.* 1.

1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 22, Cadogan gardens, S.W.

1882 WHITE, EDWIN FRANCIS, Westlands, 280, Upper Richmond Road, Putney, S.W.

1890 WHITE, GILBERT B. MOWER, M.B., B.S., 112, Harley Street, W.

1883 WHITE, WILLIAM HALE, M.D. 65, Harley Street, W. (C. 1887–9, 1896–9, S. June, 1893–6, V.P. 1906–7. V.-P. Clin. Sect. Roy. Soc. Med., 1907– .) *Trans.* 17, *C.C.* 4.

1883 WHITE, WILLIAM HENRY, M.D., 43, Weymouth Street, W. *Trans.* 1. *C.C.* 1.

1894 WHITELOCKE, RICHARD HENRY ANGLIN, M.B., C.M., 6, Banbury Road, Oxford.

1897 WHITFIELD, ARTHUR, M.D., 21, Bentinck Street, Manchester Square, W.

1882 WHITTLE, EDWARD GEORGE, M.D., 9, Regency Square, Brighton.

1871 WIGHT, GEORGE, M.B., C.M., 428, Liverpool Road, N.

ELECTED

1879 WILCOX, HENRY, M.B., Newlyn, Fleet, Hants.
1894 **WILKIN**, GRIFFITH CHARLES, Conway House, Paignton, South Devon.
O.M. WILLETT, ALFRED, 26, Upper Wimpole Street, W. (C. 1872–5, V.P. 1889–91.) *C.C.* 2.
1888 WILLIAMS, CAMPBELL, 18, Queen Anne Street, Cavendish Square, W. *Trans.* 2.
O.M. WILLIAMS, CHARLES THEODORE, M.V.O., M.D., 2, Upper Brook Street, W. (C. 1877–9, V.P. 1889–90.) *Trans.* 8.
1888 WILLIAMS, DAWSON, M.D., 2, Agar Street, Strand, W.C. (C. 1893–7.)
1903 WILLIAMS, LEONARD, M.D., 8, York Street, Portman Square, W. *C.C.* 1.
1890 WILLIAMS, W. ROGER, Beaufort House, Clifton Down, Clifton, Bristol.
1893 WILLS, JOSEPH PEARCE BUDGETT, M.D., Chintings, Bexhill-on-Sea.
1888 Wilson, CLAUDE, M.D., C.M., Belmont, Tunbridge Wells. (C. 1905–7. C. Clin. Sect. Roy. Soc. Med., 1907– .)
1891 WILSON, THEODORE STACEY, M.D., C.M., 27, Wheeley's Road, Edgbaston, Birmingham. *Trans.* 2.
1905 WIRGMAN, C. WYNN, M.D., B.S., 45, Queen Anne Street, Cavendish Square, W.
1879 WOODWARD, GEORGE P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.
1894 WOOLLETT, CHARLES JEROME, "Ambleside," Streatham, S.W.
1884 WORTS, EDWIN, 6, Trinity Street, Colchester.
1892 WYNTER, WALTER ESSEX, M.D., B.S., 27, Wimpole Street, Cavendish Square, W. *C.C.* 4.
1872 YEO, I. BURNEY, M.D., 44, Hertford Street, W. (C. 1881–3.) *Trans.* 8.

LIST OF MEMBERS.

ORIGINAL MEMBERS (ALPHABETICALLY).

Henry Arnott.
Richard Barwell.
Henry Charlton Bastian, M.D., F.R.S.
Thomas Bryant.
Thomas Buzzard, M.D.
Sir William Selby Church, Bart., K.C.B., M.D.
Edward Clapton, M.D.
John Couper.
William Howship Dickinson, M.D.
Sir Dyce Duckworth, M.D.
Jonathan Hutchinson, F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
John Langton.
Arthur Trehern Norton, C.B.
Frederick William Pavy, M.D., F.R.S.
Sir Richard Douglas Powell, Bart., K.C.V.O., M.D.
Sydney Ringer, M.D., F.R.S.
Sir Thomas Smith, Bart., K.C.V.O.
Sir Hermann D. Weber, M.D.
Alfred Willett.
Charles Theodore Williams, M.D.

ARRANGED ACCORDING TO DATE OF ELECTION.

1868 Sir Constantine Holman, M.D.
Christian G. H. Bäumler, M.D.
Howard Marsh, M.C.
Charles Prentis.
Sir Edgcombe Venning.
John Ford Anderson, M.D.
George Granville Bantock, M.D.
George Charles Bright, M.D.
Frank W. Cooper.
Edward Hooper May, M.D.
William Warwick Wagstaffe.
James Edward Pollock, M.D.
Franz Oppert, M.D.
William V. Snow, M.D.
Charles Drage, M.D.
Frederick Royston Fairbank, M.D.
Michael W. Rice, M.D.

1869 J. Warrington Haward.
Henry Frederick Augustus Goodridge, M.D.
Olliver Thomas Duke, M.B.

1871 Alfred Playne, M.B.
George Wight, M.B.
Ebenezer Diver, M.D.

1872 I. Burney Yeo, M.D.
Henry Harris, M.D.
William Pugin Thornton.
Sir G. Anderson Critchett.
J. C. J. Fenwick, M.D.
Andrew S. Myrtle, M.D.
Sir William Bartlett Dalby, M.B.
Thomas Ridge Jones, M.D.
George Slight, M.D.

1873 David Lloyd Roberts, M.D.
George Henry Savage, M.D.

1873 Edwin Chisholm, M.D.
Thomas Churton, M.D.
1874 John Hammond Morgan, C.V.O.
Edward R. Rowland.
Claudius Galen Wheelhouse.
Edward C. Stirling, M.D.
Sir William Henry Bennett, K.C.V.O.
William Brown Holderness.
Andrew Clark.
1875 Sir Thomas Barlow, Bart., K.C.V.O., M.D.
Sidney Coupland, M.D.
Clinton T. Dent.
C. D. B. Hale.
Rickman John Godlee, M.S.
Arthur Paul Sherwood.
James Frederic Goodhart, M.D.
Sir William Richard Gowers, M.D., F.R.S.
William Smith Greenfield, M.D.
Sir Shirley F. Murphy.
Herbert W. Page.
Frederick Taylor, M.D.
1876 Arthur E. J. Barker.
Horatio Percy Symonds.
A. Balmanno Squire.
David White Finlay, M.D.
Furneaux Jordan.
R. Clement Lucas, B.S.
George Buckston Browne.
Arthur Edwin Temple Longhurst, M.D.
1877 Robert Hogarth Clay, M.D.
A. Pearce Gould, M.S.
Henry Radcliffe-Crocker, M.D.
David B. Lees, M.D.
William Ewart, M.D.
Henry Morris, M.B.
Henry Ambrose Lediard, M.D.
Bernard Roth.
Henry Hugh Clutton, M.C.
Malcolm Alex. Morris.
1878 George P. Field.
Thomas Warner Lacey.
Thomas Colcott Fox, M.B.
Sir Felix Semon, C.V.O., M.D.
Henry de Fonmartin, M.D.
C. H. Golding-Bird, M.B.
Donald William Charles Hood, M.D.
Lord Lister, P.C., O.M., F.R.S.
F. de Havilland Hall, M.D.
William Allen Sturge, M.D.
1878 William Joseph Tyson, M.D.
Charles Robert Bell Keetley.
William Appleton Meredith, C.M.
Robert Bridges, M.B., M.A.
Frederick William Strugnell.
1879 William Edward Burton.
James Magill, M.D.
Henry Wilcox, M.B.
Sir Stephen Mackenzie, M.D.
William Harrison Cripps.
Francis Patrick Staples.
Geo. Courtenay Henderson, M.D.
Henry Davy.
Thos. Walter Harropp Garstang.
Charles W. Mansell Moullin.
John Reuben Lunn.
George P. M. Woodward, M.D.
Robert Peel.
Frederic S. Dennis, M.D.
1880 T. Mark Hovell.
Henry Francis Baker.
Bernard O'Connor, M.D.
Charles Edward Beevor, M.D.
1881 George Henry Makins, C.B.
James Kingston Fowler, M.D.
Charles Edward Harrison, M.B.
Malcolm Macdonald McHardy.
Rushton Parker.
Montagu Lubbock, M.D.
William Pasteur, M.D.
John Caldwell Uhthoff, M.D.
Henry Trentham Butlin.
H. A. Powell, M.A.
1882 George Robertson Turner.
Robert William Goldie.
Frederick Charles Barker, M.D.
Daniel Colquhoun, M.D.
Seymour Taylor, M.D.
Philip Henry Bindley, M.B.
Edward George Whittle, M.D.
Frederick Henry Spooner, M.D.
Frederick Haycraft Berry, M.D.
Herbert Collier, M.D.
Samuel West, M.D.
Emile Emond, M.D.
Charters James Symonds, M.S.
C. F. Coxwell, M.B.
Augustus Joseph Pepper, M.B.
Edwin Francis White.
1883 Anthony A. Bowlby, C.M.G.
Percy Kidd, M.D.
William Henry White, M.D.
Hubert Montague Murray, M.D.

1883 Robert Fitzroy Benham.
William Henry Allchin, M.D.
John Mitchell Bruce, M.D.
William Arbuthnot Lane, M.S.
William Hale White, M.D.
William Coode Adams, M.B.
Robert Leamon Bowles, M.D.
George Henry Jackson.
Thomas Laurence Read.
Frederick Thomas Roberts, M.D.
Charles Alfred Ballance, M.B., M.S.

1884 R. Percy Smith, M.D.
Edgar Duke.
John Mackern, M.B.
Paul M. Chapman, M.D.
Wilmot Parker Herringham, M.D.
Philip Henry Pye-Smith, M.D., F.R.S.
Charles Stonham, C.M.G.
Dudley Wilmot Buxton, M.D.
Edwin Worts.
Seymour J. Sharkey, M.D.
John James Pringle, M.B.
Frederick Lucas Benham, M.D.
Walter Edmunds, M.C.
Stephen Paget.
Sir Lambert Hepenstal Ormsby, M.D.
Edwin Leonard Adeney, M.D.
Sir Victor Horsley, F.R.S.
Henry Carr Maudsley, M.D.
Bilton Pollard, B.S.

1885 Frederick Spicer, M.B.
James Berry, B.S.
Fred. Walker Mott, M.D., F.R.S.
George Newton Pitt, M.D.
W. C. Everley Taylor.
Sidney Phillips, M.D.
A. W. Mayo Robson.
Thomas Wakley, jun.
Thomas William Thursfield, M.D.
Alexander Dalton Murray, M.B.
Robert Maguire, M.D.
Robert Alexander Gibbons, M.D.
Henry Brewer Tait.
Richard Caton, M.D.
Arthur Henry Robinson, M.D.
Edward Sabine Tait, M.B.
William Bruce Clarke, M.B.
Charles Barrett Lockwood.
Reginald J. Ryle, M.D.

1885 J. Michell Clarke, M.D.
Henry George Armstrong.
Roderick Maclaren, M.D.
W. Watson Cheyne, C.B., F.R.S.

1886 Thomas Dixon Savill, M.D.
Benjamin Wainewright, M.B.
Waren Tay.
William Henry Battle.
James Hardie, M.D.
Francis Henry Hawkins, M.D.
R. Hingston Fox, M.D.
John Ward Cousins, M.D.
T. Pridgin Teale, F.R.S.
H. Lankester, M.D.
Charles Herbert Thompson, M.D.
Henry Handford, M.D.
Alfred Scott.

1887 Archibald E. Garrod, M.D.
H. T. Rutherfoord, M.B.
Kankai Totsuka.
Thomas Oliver, M.D.
Samuel Herbert Habershon, M.D.
Oswald Auchinleck Browne, M.D.
Joseph Arderne Ormerod, M.D.
J. H. E. Brock, M.D., B.S.
Francis William Clark.
E. Hurry Fenwick.
R. Lawford Knaggs, B.C.
John D. Malcolm, M.B., C.M.
Sidney Martin, M.D., B.S., F.R.S.
Thomas Horrocks Openshaw, C.M.G., M.B.

1888 A. G. Barrs, M.D.
J. W. Batterham, M.B., B.S.
Montagu Handfield-Jones, M.D.
Robert Henry Scanes Spicer, M.D.
Campbell Williams.
Frederic S. Eve.
Alexander Morison, M.D.
Frederick Page, M.D.
Frederick J. Smith, M.D.
Frederick R. Walters, M.D.
Claude Wilson, M.D., C.M.
Charles H. Gage-Brown, M.D.
J. H. Menzies.
Frank Ernest Roberts.
Hugh Armstrong.
Hyde Marriott, M.B.
Percy Warner.
J. T. James, M.D.
Edwin A. Barton.
W. Page May, M.D.

1888 Philip D. Turner, M.D.
Dawson Williams, M.D.
Augustus W. Addinsell, M.B., C.M.
John Anderson, C.I.E., M.D.
Henry French Banham, M.D.
David James Mason, M.D., C.M.
Walter G. Spencer, M.B., M.S.

1889 Theodore Dyke Acland, M.D.
Raymond Johnson, M.B., B.S.
H. D. Rolleston, M.A., M.D.
P. MacBride, M.D.
D. Newman, M.D.
Herbert Elwin Harris, M.B.
John E. Ranking, M.D.
Stanley Boyd, B.S.
George Ezra Halstead, M.D., B.S.
Henry Herbert Taylor.
John Duncan, M.D.
Wm. Wallis Ord, M.D., B.Ch.
Leonard Arthur Bidwell.
Arthur J. M. Bentley, M.D.
Francis R. B. Bisshopp, M.D., B.Sc.
Henry Percy Dean, M.B.
Louis Albert Dunn, M.S.
Herbert Pennell Hawkins, M.D., B.Ch.
Lauriston Elgie Shaw, M.D.

1890 John Rose Bradford, M.D., F.R.S.
J. Fletcher Little, M.B.
Robert Robertson, M.D.
Ernest Solly, M.B.
James Taylor, M.D.
Francis O. Buckland, B.A., M.B. C.M.
Edmund W. Roughton, B.S.
James Calvert, M.D.
H. Roxburgh Fuller, M.D.
Arthur F. Voelcker, M.D.
W. Roger Williams.
Gilbert B. M. White, M.B., B.S.
Frederick Charles Wallis, M.B., B.C.
Alfred Parkin, M.S.
George A. Hawkins-Ambler.
James Harper, M.D.
Walter Henry Brown.
John Walter Carr, M.D.
Ernest Le Cronier Lancaster, M.B., B.Ch.
Sir Patrick Manson, K.C.M.G., M.D., F.R.S.

1890 Miles Miley, M.A., M.B.
Edgar Herbert Thane, M.B.
Charles William Chapman, M.D.
Michael G. Foster, M.A., M.D.

1891 Frederic François Burghard, M.D., M.S.
Roger Neville Goodman, M.B.
Herbert Furnivall Waterhouse, M.D., C.M.
Walter William Hunt Tate, M.D.
Greville MacDonald, M.D.
J. Kingston Barton.
J. Dundas Grant, M.D.
W. Kington Fyffe, M.B., B.C.
Albert Carless, M.B., M.S.
W. Bolton Tomson, M.D.
Harry Littlewood, M.D.
Hector W. G. Mackenzie, M.A., M.D.
Chas. Percival White, M.B., B.C.
Arnold Caddy.
Theodore Stacey Wilson, M.D., C.M.

1892 William Hunter, M.D.
Frank Thomas Paul.
Walter Essex Wynter, M.D., B.S.
Damer Harrisson.
John Alfred Masters, M.D.
Walter Gifford Nash.
John Stephenson Selwyn-Harvey, M.D.
Edward John Lewis, M.B., B.C.
Henry Betham Robinson, M.D. M.S.
Richard James Herbert Scott.
Howard Henry Tooth, C.M.G. M.D.

1893 John Ernest Paul, M.B.
James William Bond, M.D.
Harry Campbell, M.D.
W. Soltau Fenwick, M.D.
Ernest Alfred Snape, M.D.
Lewis G. Glover, M.B., B.C.
William Bracewell Mercer, M.B., B.C.
Robert Henry Cole, M.D.
Donald Rose Paterson, M.D., C.M.
Walter Spencer Anderson Griffith, M.D.
Alfred Theodore Rake, M.B., B.S.
George Redmayne Murray, M.D., M.A.
R. J. Bliss Howard, M.D.

1893 Henry Jones Curtis, M.D., B.S.
Henry Albert Caley, M.D.
Theodore Henry Ionides, M.B., B.S.
Arthur Foxwell, M.D.
Horace George Turney, M.D.
Henry Grabham Lys, M.D.
G. F. Johnston, M.D.
Willmott H. Evans, M.D., B.S.
Charles Gibbs.
Robert Stephen Charsley.
Joseph Pearce Budgett Wills, M.D.
Arnold Lawson, M.D.

1894 Herbert Morley Fletcher, M.D.
Edward Wilberforce Goodall, M.D.
Harry Gilbert Barling.
Ernst Michels, M.D.
Charles Jerome Woollett.
StClair Thomson, M.D.
George A. Sutherland, M.D.
John Murray.
Richard Henry Anglin Whitelocke, M.B., C.M.
John Attlee, M.D., B.C.
Griffith Charles Wilkin.
Thomas Hugh Dickson, M.B., B.C.
C. Gordon Brodie.
Bernard Scott.
John Porter Parkinson, M.D.
Thomas Rudolph Smith, M.B., B.C.
Arthur Pearson Luff, M.D.

1895 Herbert Murray Ramsay.
Arthur G. Phear, M.D., B.C.
Ernest E. Lewis, M.D.
Karl Grube, M.D.
Gerald R. Baldwin.
James Galloway, M.D.
F. B. Willmer Phillips, M.B.
Charles Henry Hough.
Leonard George Guthrie, M.D.
James Ernest Lane.
Frank J. Wethered, M.D.
Richard Ackerley.

1896 Charles Spurrell.
Cuthbert Sidney Wallace, M.B.
Charles R. Box, M.D.
Walter Stacy Colman, M.D.
Edward Ward, M.B.
Norman Dalton, M.D.
Fred. Parkes Weber, M.D.
Francis James Steward, M.B., B.S.

1896 William Bezly Thorne, M.D.
Douglas Drew, M.D., B.S.
Surg.-Capt. W. R. Crooke-Lawless, A.M.S., M.D.
William Elliot Risdon, M.D.
Priestley Leech, M.D.
Ebenezer Stanley Smith, M.D.
Edward Cooper Bentham.
Robert Herbert Mills-Roberts, C.M.G.
Jonathan Hutchinson, jun.
Frederick E. Batten, M.D.
Rutherford John Pye-Smith.
George Templeton.
Bertram Louis Abrahams, M.B., B.Sc.

1897 Raymond H. P. Crawfurd, M.D.
John Henry Deane.
Percy Furnivall.
Arthur Whitfield, M.D.
Frederick William Forbes Ross, M.D.
Harold Low, M.B.
Charles Ralph Keyser.
George Bertram Hunt, M.B.
Alfred Herbert Tubby, M.S.
Charles Lamplough.
H. Campbell Thomson, M.D.
E. Claude Taylor, M.D.
Charles Humphrys.
Charles Sempill de Segundo, M.B.
Sir Alfred Downing Fripp, C.B., M.V.O., M.S.
Chichester Gould May, M.D.

1898 J. J. Perkins, M.D.
James Jackson Clarke, M.B.
Ernest Herbert Cobb.
Sydney Backhouse Hulke.
Thomas Herbert Kellock.
William Darley-Hartley.
Robert Jones.
Arthur Philip Beddard, M.D.
Alfred Ernest Russell, M.D., B.S.
William Cecil Bosanquet, M.D.
Arthur A. H. Partridge, M.B., M.Ch.
Leonard Avery.
William Turner, M.B., B.S.
Harold Batty Shaw, M.D.
Joseph Stewart Richards, M.D., B.S.
William Henry Breffit Brook, M.D., B.S.

1899 George Edward Rennie, M.D.
V. Warren Low, M.D., B.S.
Rayner Derry Batten, M.D., B.S.
Charles E. Forbes Mouat-Biggs.
Rutherford Morison.
R. Murray Leslie, M.D.
Herbert Tilley, M.D.
Charles Owen Fowler, M.D.
Cyril Ogle, M.B.
Charles Herbert Fagge, M.S.
Claude Somerville Hawkes.
Henry Stephen Sandifer, M.D., B.S.
Marcus Marwood Bowlan, M.B., B.Sc.
Alfred Eïd, M.D.
Karl Fürth, M.D.
Wm. Gream Stone, M.D.
Andrew Duncan, M.D.
Edmond Lucien De Chazal, M.D.
Henry Roe Walker.
Edward Collingwood Andrews, M.D.

1900 Berkeley George Andrew Moynihan, M.S.
Harry Cooper, M.D.
George Augustus Tullis, M.D.
Griffith Lloyd Roberts, M.B., C.M.
Edward Percy Paton, M.S.
John Arthur Hayward, M.D.
Wilfred J. Hadley, M.D.
John Fawcett, M.D.
Arthur Bousfield, M.B.
J. S. Risien Russell, M.D.
Cecil H. Leaf, M.B.
Josef Paul zum Busch, M.D.

1901 Ernest George Leopold Goffe, M.D.
Arthur Hamilton Nicholson Lewers, M.D.
Charles Reissmann, M.D.
Adrian Caddy, M.B.
Herbert William Carson.
Thomas Henry Morse.
Laurie Hugh McGavin.
F. U. Menzies.
Thomas Rupert Hampden Bucknall, M.S.
Edward Angas Johnson, M.B.
Percival Horton-Smith Hartley, M.D.

1901 Sir Hugh Reeve Beevor, Bart., M.D
Theo. N. Kelynack, M.D.
Thomas Carwardine, M.S.
Bowie Campbell Gowan.
John J. Douglas, M.D.
Harold Shuttleworth Barwell.

1902 Charles Henry Fennell, M.D.
Harold L. Barnard
Thomas Percy Legg, M.B.
Andrew Elliot, M.D.
Sir James Reid, Bart., G.C.V.O., K.C.B.
F. W. Price, M.B.
Alfred Ernest Sears.
Reginald F. Jowers.
Cecil Marriott, M.B.
Edward Farquhar Buzzard, M.D.
Donald John Armour, M.B.
Edred Moss Corner, B.Sc., M.B.
Herbert French, M.D.
Lawrence Jones, M.B.
Thomas Crisp English.
Walter Fedde Fedden, M.B.
David Nabarro, M.D.
Charles A. Morton.
Victor Evelyn Collins, M.B.
Leonard S. Dudgeon.
R. O. Moon, M.D.
Charles Gordon Watson.
Charles Firmin Cuthbert.

1903 Raoul Ernest Delbruck, M.B.
Edward A. Gates, M.D.
E. Graham Little, M.D.
Percy Robert Cooper, M.D., B.Sc.
Leonard Williams, M.D.
Ernest Lloyd Jones, M.D.
John Basil Hall, M.C.
Thomas Reuell Atkinson, M.D.
Graham Simpson.
James William Russell, M.D.
Philip Maynard Heath, M.B.
Wilfred Harris, M.D.
Arthur S. Morley.
Hugh Rigby, M.S.

1904 Holburt J. Waring, M.S.
Charles Bolton, M.D.
R. P. Rowlands, M.S.
H. A. Ballance, M.S.
Frederic W. Longhurst.
H. S. D. Browne, M.B., B.C.
Philip Turner, M.S.
George Herbert Graham, M.D.

1904 George Grey Turner, M.B., M.S.
John English Harburn.
Oswald Lacy Addison, M.B.
A. Ernest Jones, M.D., B.Sc.
1905 Harold A. T. Fairbank, M.S.
David Forsyth, M.D.
E. Rock Carling, M.B., B.S.
Joseph Cunning, M.B., B.S.
William Henry Bailey, M.D., D.P.H.
James Mear Brydone, M.B., B.C
Francis Frederick Muecke, M.B., B.S.
William Frederick Brook
1905 Joseph Arthur Arkwright, M.D., B.C.
William Henry Blake, M.D.
John Paul Roughton.
C. Wynn Wirgman, M.D., B.S.
1906 Cecil Rowntree, B.S.
Reginald George Hann.
John Roger Charles, M.D.
J. H. Drysdale, M.D.
James Sherren.
Charles Ryall.
John Seymour Maynard, M.D.
1907 H. Gwynne Lawrence, M.B.
Henry Percival Johnson.
Cecil Irving Graham.

THE CLINICAL SOCIETY OF LONDON.

Statement of Cash Receipts and Payments from the 1st May, 1906, to the 30th April, 1907.
G. H. MAKINS, Esq., C.B., *Treasurer.*

RECEIPTS.	£	s.	d.
Balance, 1st May, 1906	45	10	10
166 Subscriptions at £1 1s. received by Collector — £174 6 0			
154 Subscriptions at £1 1s. received by Bank — 161 14 0			
320	336	0	0
39 Subscriptions at 10s. 6d. received by Collector — 20 9 6			
1 Subscription at 10s. 6d. received by Bank — 0 10 6			
	21	0	0
3 Admission Fees at £2 2s.	6	6	0
1 Composition Fee	4	4	0
Sale of *Transactions*	11	11	2
Proceeds of Sale of Consols, £68 18s. 2d.	59	9	0
Dividend on Consols	12	2	2
Remission of Income Tax	2	6	8
Per Dr. L. H. Marshall (paid in error and since refunded)	2	0	0
	£500	9	10

Amount invested in Consols, £600.

PAYMENTS.		£	s.	d.
Cost of *Transactions*, Vol. XXXIX:				
Printing, paper, delivery, &c.	£142 6 2			
Illustrations	47 9 0			
		189	15	2
Meetings:				
Rent of Rooms	105 0 0			
Hire of Microscopes and Lanterns	3 2 0			
Hire of Epidiascope	6 10 0			
Share of expenses incurred by Executive Committee in promoting union of Medical Societies	6 6 8			
A. Tapson (attendance)	7 10 0			
Refreshments	33 8 0			
		161	16	8
Treasurer and Hon. Secretaries:				
Secretarial assistance	26 5 0			
Commission to Collector	15 15 0			
		42	0	0
Printing and Stationery		75	15	5
Fees for nominal transfer of Consols		1	0	3
Bank Charges		2	12	6
Petty Cash Expenditure		10	0	6
Balance at Bank	13 10 7			
Petty Cash	1 18 9			
		15	9	4
Cheque refunded to Dr. Marshall		2	0	0
		£500	9	10

H. H. CLUTTON, *President.*
G. H. MAKINS, *Hon. Treasurer.*
WALTER G. SPENCER, *Hon. Secretary.*

Audited and found correct,
J. WALTER CARR, M.D.,
F. F. BURGHARD, } *Auditors.*
May 14th, 1907.

REPORT

OF THE

COUNCIL OF THE CLINICAL SOCIETY,

MAY, 1907.

WITH the conclusion of the present session the Clinical Society ceases to exist as a distinct Society, and will become merged in the new Royal Society of Medicine.

Founded in the year 1867, with Sir Thomas Watson as its first President, the Clinical Society can look back upon forty years of useful work, and may regard with pride the list of highly distinguished men who have filled its Presidential chair, and who have contributed to the forty volumes of its 'Transactions.' The 'Transactions' contain a large mass of valuable clinical records, the usefulness of which will long outlast the existence of the Society, and one of the last acts of its Council has been to render them easily available for reference by arranging for the completion of the general index.

Committees appointed by the Society have done much valuable work, and the supplementary volumes on the 'Incubation Periods of Infectious Diseases' and on 'Myxœdema' are worthy of special mention. Myxœdema is, indeed, a malady which will always remain closely associated with the name of the Clinical Society, before which it was first described in Sir William Gull's classical paper.

We may look forward with confidence to the continuation of the work of the Society on the same lines as heretofore by the Clinical Section of the Royal Society of Medicine.

At a Special General Meeting of the Society on November 23rd, 1906, the following resolution of the Council of November 9th was read:

"That the Council of the Clinical Society, being satisfied that the scheme of Amalgamation of the Medical Societies of London is financially feasible, does advise the Clinical Society to agree to form the said Amalgamation, and suggest to the Society the name of Mr. Makins as its delegate to the new Committee."

The adoption of the above resolution was proposed by Dr. Hale White, Vice-President; seconded by Dr. F. Taylor, Past President; spoken to by Mr. J. H. Morgan, Trustee; and unanimously agreed to.

The Society numbers 17 Honorary Members and 572 Ordinary Members. During the past session 5 new members have been elected to, and 14 have resigned from the Society.

The Society has lost by death 6 members: Dr. A. H. W. Clemow, Mr. E. A. Fardon, Mr. D. H. Goodsall, Mr. J. F. C. H. Macready, Dr. G. Schorstein, Dr. F. Willis.

Minutes of Special General Meeting held June 14th, 1907.

A Special Meeting of the Clinical Society was held at 20, Hanover Square, on Friday evening, June 14th, 1907, at 8.30 p.m.

Present twenty-two members.

The President, Mr. H. H. Clutton, in the chair.

The minutes of the last meeting were read and confirmed.

The following resolutions were submitted by the senior Secretary, and their adoption was proposed by Sir Dyce Duckworth and seconded by Dr. Pasteur:

1. "Resolved that, in pursuance of the decision of the Society on November 23rd, 1906, to unite with other societies in forming the Royal Society of Medicine, which has now received a Royal Charter, all the property, of every kind whatsoever, of this Society be and is hereby transferred to the Royal Society of Medicine, and the Treasurer and Trustees are hereby directed forthwith to transfer to the Royal Society of Medicine all sums of money and all invested funds in their possession standing in their names or at the credit of the Society at its bank."

2. "Resolved, that the Clinical Society of London, in handing over its 'Archives' to the Royal Society of Medicine, expresses a wish that these 'Archives' may be entrusted to the keeping of the Clinical Section of the Royal Society of Medicine."
3. "Resolved, that the Clinical Society of London be discontinued, and is hereby dissolved."

The resolutions were voted upon in turn, and each was carried unanimously.

H. H. Clutton.
Walter G. Spencer.
Archibald E. Garrod.

Minutes relating to the Letter of Congratulation sent to Lord Lister.

At a meeting of the Clinical Society, held at 20, Hanover Square, on Friday evening, April 12th, 1907, Mr. Charters Symonds, Vice-President, in the chair, the Chairman read a draft prepared by the Council of a letter to Lord Lister, Past President of the Society, congratulating him on the eightieth anniversary of his birth. Having been submitted from the Chair, the form of the proposed letter was approved by the Society, and it was arranged that it should be signed by the President and Honorary Secretaries and forwarded to Lord Lister.

At a meeting of the Clinical Society, held at 20, Hanover Square, on Friday evening, May 10th, 1907, Mr. H. H. Clutton, President, in the chair, the President read a reply from Lord Lister to the letter of congratulation sent to him on behalf of the Society. The reply was as follows:

(*See next page.*)

12, PARK CRESCENT,
PORTLAND PLACE.

19 April 07

My dear Mr Clutton

I beg to thank
you and the Hon Secretaries
very warmly for your
most kind letter of
congratulation

I look back upon the
period of my Presidency of
the Clinical Society with
peculiar pleasure.

Believe me
very sincerely Yours,

Lister

PRESIDENTIAL ADDRESS
ON ADOLESCENT OR LATE RICKETS

DELIVERED AT THE

CLINICAL SOCIETY OF LONDON

ON OCTOBER 12TH, 1906.

BY

H. H. CLUTTON, M.A., M.C.CANTAB., F.R.C.S.ENG.,

PRESIDENT.

With Plates I—IV.

GENTLEMEN,—I beg to welcome you to this Society for another session and hope that much pleasure and profit may accrue to those who take part in our deliberations. I cannot help wishing that more men would come and discuss the cases that are presented to the Society, for I feel confident that in attempting to correct others in their errors we end in correcting ourselves. It is very curious to notice the number of members who come to see patients on the clinical evenings and how few of them remain to ask questions of, or to criticise the opinions of, the exhibitors. Is it possible that in the hurry of work men no longer wish to discuss their cases? I should be sorry to think that they have lost interest in clinical medicine and surgery, for there must always be two sides, if not more, in every question of difficult diagnosis. It is customary, as you know, for the President, when elected for a second year of office, to give an address. First, let me thank you for giving me the opportunity of assisting to make our meetings successful. Secondly, forgive me for having taken the liberty of somewhat departing from orthodox lines by starting a discussion instead of giving an address on general principles. However, I hope the subject which I have chosen will be interesting to physicians and surgeons alike.

Attention was first called in England to the subject of late or adolescent rickets by Dr. F. G. D. Drewitt, who showed a boy, æt. 10, at the Pathological Society of London in November, 1880. The boy, who was under the care of Dr. W. B. Cheadle at the Children's Hospital, died two years afterwards, and the *post-mortem* examination, made by Sir Thomas Barlow and Dr. John Abercrombie, showed that the disease was undoubtedly one of true rickets. A few cases have been recorded since then, but in some of them a confusion seems to have arisen in the diagnosis between rickets and osteomalacia. I do not wish to argue about any individual case previously recorded, but rather to proceed with those which I have in hand. It is of some importance that we should be able to assert without fear of contradiction that rickets may appear during the adolescent period of life, for it is not so long ago since this possibility was either ignored or denied. The importance of being able to ascribe a certain proportion of bony deformities that are seen in adult life to their true cause will be obvious to us all. I am not now wishing to assert that such and such cases of adult deformity are due to adolescent rickets, but rather to show what rickets can produce in the way of deformity during the adolescent period of life, and, if unrelieved, the possible deformity in the adult skeleton when all signs of the original cause—namely rickets—have disappeared. The majority of the cases of coxa vara, genu valgum, and allied cases, which are ordinarily put down to static deformity of adolescence, are in reality due to the same kind of imperfect ossification which occurs in rickets. But very few indeed show the same condition at the epiphysial lines which is seen in infantile rickets. Hence they are not usually ascribed definitely to this disease. I imagine the difference is only one of degree. The faulty metabolism which in its milder form may produce a soft bone, which yields to the weight of the body, may in a more severe degree result in a greater and more permanent change in the bony framework of the body. In the former case the bones are not sufficiently ossified, and yield such deformities as coxa vara, genu valgum, and bow legs, but the epiphysial lines do not show the proliferation of unossified cartilage which we recognise as characteristic of rickets. When, however, the epiphyses do show this condition there is little or no difficulty in stating that the case is one of rickets, and as a result we may expect to find, not only the same deformities already mentioned, but also, after a

Mali

FIG. 2.

FIG. 1.

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PLATE I.

Illustrating Mr. Clutton's Cases of Adolescent Rickets.

FIG. 1.—Photograph of Case 1.

FIG. 2.—Radiograph from above case showing curves at the knee.

prolonged continuance of the same condition at the epiphysial lines, some of the extraordinary deformities seen in adult life. The object of this paper, therefore, is to show that all the ordinary signs of infantile rickets, so far as regards the bones, may exist in the adolescent period of life, and that they may continue for many years. The history of the patient may prove either that rickets did not exist in childhood, or, assuming the history in some cases to be imperfect, that rickets must be said in the latter event to have continued from infancy to adult life, or to have been in abeyance for a time and to have reappeared at the age of puberty, for in the case I am recording at the age of twenty-one years we have, I think, positive evidence of the exact condition of the bones by means of X rays. It was not possible before the discovery of these rays—except in the rare occasion of a *post-mortem* examination—to prove the presence of active rickets in the epiphyses. Consequently the case I first recorded under the name of late rickets in 1884 lacked the confirmation I have been able to obtain in the present cases.

Case 1 (Pl. I, fig. 1).—The patient was sent to me by Mr. A. C. Robinson, and was admitted into St. Thomas's Hospital under my care in February, 1904. The history taken by my dresser, Mr. J. H. D. Acland, was as follows: The patient, æt. 21, was of Italian parentage on his father's side, and the second child of a family of five, none of whom were the subject of rickets in infancy. Dentition was not delayed and he began to walk at the usual time. There was no enlargement or curvature of the long bones during childhood. Between the ages of eleven and twelve years the patient was first noticed by his parents to walk badly. He was at that time taken to one of the orthopædic hospitals, where he was ordered steel supports for genu valgum of both limbs. These he wore for two years without any apparent improvement. He was then taken to Guy's Hospital—about five years ago, at the age of sixteen years—where osteotomy of both femora was performed by Mr. J. N. C. Davies-Colley, and plaster-of-Paris was applied for three months. After this treatment the lower limbs were quite straight and remained so for three years—*i. e.* till two years ago. At this time, at the age of nineteen years, the left femur was noticed to be bending again, and an "iron" was ordered, which seemed to correct the deformity. About nine months ago, at the age of twenty years, the legs began to drop backwards at the knees. The

right leg, which had no support, rapidly showed the greater deformity. His parents were not poor people, so that he had always been well fed. There is one point in his history which should be mentioned here, as it illustrates one of the possible contributory causes for this trouble. His school life had been a hard one. He had had to walk three miles from his home to school and three miles back. He had worked at night till eleven o'clock, and had gained a scholarship by examination as a result. He left school at sixteen years of age, and became a clerk in the City, working at a desk from 10 a.m. to 6 or 8 p.m. His young life had therefore been a strenuous one.

At present (February, 1904) the patient is a feeble-looking, narrow-chested youth without hair on his face, looking much younger than his age of twenty-one years. His occupation is that of a clerk in an office, where he is able to do an ordinary day's work. His weight is 6 st. 5½ lb., and his height 5 ft. ½ in.

In looking at Pl. I, fig. 1, it will be noticed that all the epiphyses appear to be larger than normal. But the most striking feature in the anatomy of the patient is the deformity at the knees, which appear at first sight to have undergone some displacement at the joints or at the epiphyses. The radiographs taken by Dr. A. H. Greg at St. Thomas's Hospital show most distinctly that the deformity is due to an antero-posterior curvature, of which the articulation is the centre, and that the bending has taken place in each bone on the diaphysial side of the epiphyses. In Pl. I, fig. 2, the combined curvatures of the femora and tibiæ give one the general impression of genu recurvatum in both lower extremities. In looking for an explanation of these curvatures it will be seen that the radiograph shows an imperfect ossification of the shafts of both femora and tibiæ. The ends of their diaphyses adjoining the epiphyses are large and bulbous, but they have only a thin shell of bone. The epiphyses themselves are not far removed from the normal condition for the age of the patient, except that the line is more distinct than it should be. Much the same condition is seen in the upper end of the left humerus, which is probably curved from muscular action. Indeed, at one spot—the apex of the curve—the cortical bone appears to be absent. There is no marked proliferation at the epiphysial lines, such as you would expect to see in the acute stage of active rickets. They would rather give one the impression

FIG. 2.

FIG. 1.

Bale and Danielsson, Ltd.

PLATE II.

Illustrating Mr. Clutton's Cases of Adolescent Rickets.

FIG. 1.—Radiograph of the wrist in Case 1.

FIG. 2.—Radiograph of the ankle in the same case.

of the acute stage having passed away, leaving behind a very feeble diaphysis at that part which immediately adjoins the epiphysis. In confirmation of this view of the case we have only to turn to the radiographs of the wrists (Pl. II, fig. 1), when we see that the epiphyses of both the long bones are in exactly the same condition as are those of young children who are the victims of active rickets. There is marked proliferation of imperfect bone with very considerable enlargement at the epiphysial line, and the adjoining diaphysis is feeble, but has no marked swelling, as is seen in the femur. The bones of the forearms have not become curved, as they have not been forced to carry weight, as in the case of the lower extremities. The skull shows no changes, probably because ossification was complete before the onset of the disease. Coxa vara does not exist, possibly for the same reason. The curvatures of the femora and tibiæ, which are so close to the epiphysial lines, suggest that imperfect ossification has been going on for some years. The result of such feeble ossification is shown in the skiagrams by the hollow shaft in the part immediately adjoining the epiphysis, and by the fact that this is the part which has yielded to the weight of the body and produced a well-marked curvature. In the case of the left humerus the curvature is a little further removed from the epiphysial line. The disease in the wrists appears, on the other hand, to be more recent and to be still in the active stage, and without such evidence as they give one might possibly doubt whether after all the curvature at the knees and shoulder was due to rickets. These beautiful skiagrams by Dr. Greg, to whom I am so much indebted, were taken in 1904, and he has been good enough to repeat the whole series in 1906. There is not much change in the bones at the knees, except that ossification is a little more complete, but at the wrists it is interesting to see a distinct improvement as regards the rachitic invasion of the diaphyses.

In a similar case, which I recorded in Vol. XIV of *St. Thomas's Hospital Reports*, 1884, the same sequence of events took place. There was a complete absence of any history of deformity or enlargement of bone-ends in early childhood, but just before the onset of puberty the wrists were for the first time noticed to be enlarged. The girl was lost sight of at the age of fourteen years, so that I am unable to report the condition of her bones at the age of twenty years. There were already some slight curvatures of the long bones in the

lower extremities at the age of twelve years, but I cannot say that they assumed the proportions later, as in the case now recorded. In 1884 we had no means, such as now exist by X rays, of knowing the exact structure of the bones.

In the present case the normal process of ossification appears to have commenced to go wrong at about the same age. Deterioration advanced still further from imperfect metabolism, continued for some years, till finally the bones were not strong enough to carry the weight of the body. This appears to have reached its climax between the ages of nineteen and twenty-one years, and the same imperfect structure exists at the present time, when the patient's age is twenty-three years. But the bones are not all in the same condition. In some of them, as, for example, at the wrists, the epiphyses of the radius and ulna showed signs of active rickets in 1904, which could scarcely be the case if the disease had been arrested. It is difficult to believe, even apart from the history, that these wrists can have been in this condition from infancy. And now, in 1906, they show some signs of recovery. In the shoulder and in the lower extremities the shafts of the long bones show less bone than usual within an attenuated cortex (Pl. II, fig. 2). This is especially the case at the parts nearest the epiphyses which have yielded to pressure. The epiphyses themselves, on the other hand, do not show any proliferation of imperfect bone, such as that seen in the radius and ulna. The present condition of these bones of the shoulder and lower extremities indicates that recovery is taking place, and that the damage done to the diaphyses has been continuous in action over many years. The contrast in this respect between some of the bones of the upper and lower extremities is very marked, and confirms, I think, the impression of the persistent character of this particular case of adolescent rickets.

Another feature of some importance should be noticed: none of these bones show any indication of sclerosis, even in those in which there is some evidence of a tendency towards recovery, nor are there any buttresses of bone thrown out on the concavity of the curvatures. In infantile rickets, on the other hand, the bones as a general rule show marked sclerotic changes after a few years, especially towards the period when recovery is taking place. Those of us who have done much osteotomy will remember the great difference there is in trying to drive a chisel or saw through the bones of different cases of rickety deformities.

An analysis of the urine that was passed during twenty-four hours was made by Dr. A. W. Crossley, consulting chemist to the hospital, on February 22, 1904. The total amount of urine was 495 c.c. It did not contain albuminoses. Its specific gravity was 1027; calcium as CaO, 0·0640 grm.; and phosphoric acid P_2O_5, 0·9595 grm. The average of calcium should be 0·30 grm., and phosphoric acid 2·5 grm. It will thus be seen that the calcium and phosphoric acid were below the normal amount. This is what one might expect in this stage of the disease, where the changes in the bone have already taken place.

Case 2.—In January, 1906, my attention was kindly called by Dr. W. S. Colman to another case, which serves as a very useful contrast to that of the patient whom I have just described. Dr. Colman's patient was a girl, æt. 13, living in Lambeth, whose history may be given as follows: She was the fourth child in a family of eight, and was breast-fed for the first fifteen months of her infant life, and for the following nine months was generally in bed with measles, bronchitis, and whooping-cough. During this time she was constantly sick and took no solid food. She may therefore be said to have had every inducement to fall a victim to infantile rickets. But so far as the history can be relied upon, there were no enlargements of bone-ends or curvatures of limbs. Nor were there any other indications of rickets, except the fact of being ill till two years of age. After this period she recovered her health and seemed like other children. In November, 1905, when she was thirteen years of age, her mother first noticed that her ankles were swollen, and in December that her knees had a peculiar shape. She was also at this time restless, irritable, and subject to profuse sweats at night. She was brought to the hospital in January, 1906, as her joints were getting worse and walking was painful. In appearance she was a healthy-looking child who showed no indication of having been infected by infantile or hereditary syphilis, and she had no albumen in her urine. With regard to hereditary syphilis, although the one existing central incisor looked at first sight suspicious, it could not be considered typical. Mr. J. H. Fisher, ophthalmic surgeon to the hospital, found no evidence of hereditary syphilis on careful examination of the eyes. On examination of the limbs she was found to have all the ordinary signs of rickets, such as you expect to see in infancy. All the long bones of

the lower extremities showed some degree of irregularity and enlargement at the epiphysial ends. Knock-knee was a conspicuous deformity, and there was a slight curvature just above each ankle. These deformities did not involve the shafts in any way, but were entirely confined to the ends of the bones. The upper extremities, with the exception of the clavicles, had no curvatures of any kind, whilst the epiphyses and the adjoining parts of the shafts were distinctly enlarged. The sternal ends of the clavicles were swollen and the natural curves were increased. The absence of curvature in the forearms, whether in the shafts or at the epiphyses, is in accordance with the generally accepted view that in infancy this occurs from crawling on the floor. In adolescence, therefore, one would not expect to see such a deformity. On careful examination of the skiagrams it will be seen that the disease is most marked on the diaphysial side of the epiphyses, that the swelling is due to a proliferation of imperfect bone at the expense of the diaphysis, and that the deviation from the straight line in the lower limbs is really the result of the want of support on the side of the epiphysis most affected. If the skiagrams of this—the younger—patient are compared with those of the older one just recorded it will be seen that the shafts of the long bones in the younger patient, æt. 13, are of normal density for this age, and are not curved, but that the deformity is confined to the junction of the epiphysis with the diaphysis. This is best seen at the ankle (Pl. III), where at first sight there would appear to be a curvature above the malleolar arch of the tibia, but the X rays show that this apparent curve is really due to a want of true bone on the inner side of the diaphysis. The skiagrams of the older patient, æt. 21, show a large hollow and curved bone for some inches above the epiphysial line. This is what one would expect if, as I am suggesting, this form of rickets arises about the age of puberty. Bone already formed would not, I imagine, be altered by this disease. In the younger subject, therefore, the shaft itself would be nearly or quite normal, whilst in the older patient, in whom the disease had existed for from seven to nine years. and who has well-marked evidences still existing at the wrists, the shaft of the long bones would be considerably altered from the prolonged imperfect ossification at the epiphysial lines, and would thereby be liable to bend from the weight of the body. It seems also as if the presence of the disease prevents the fusion which would otherwise take

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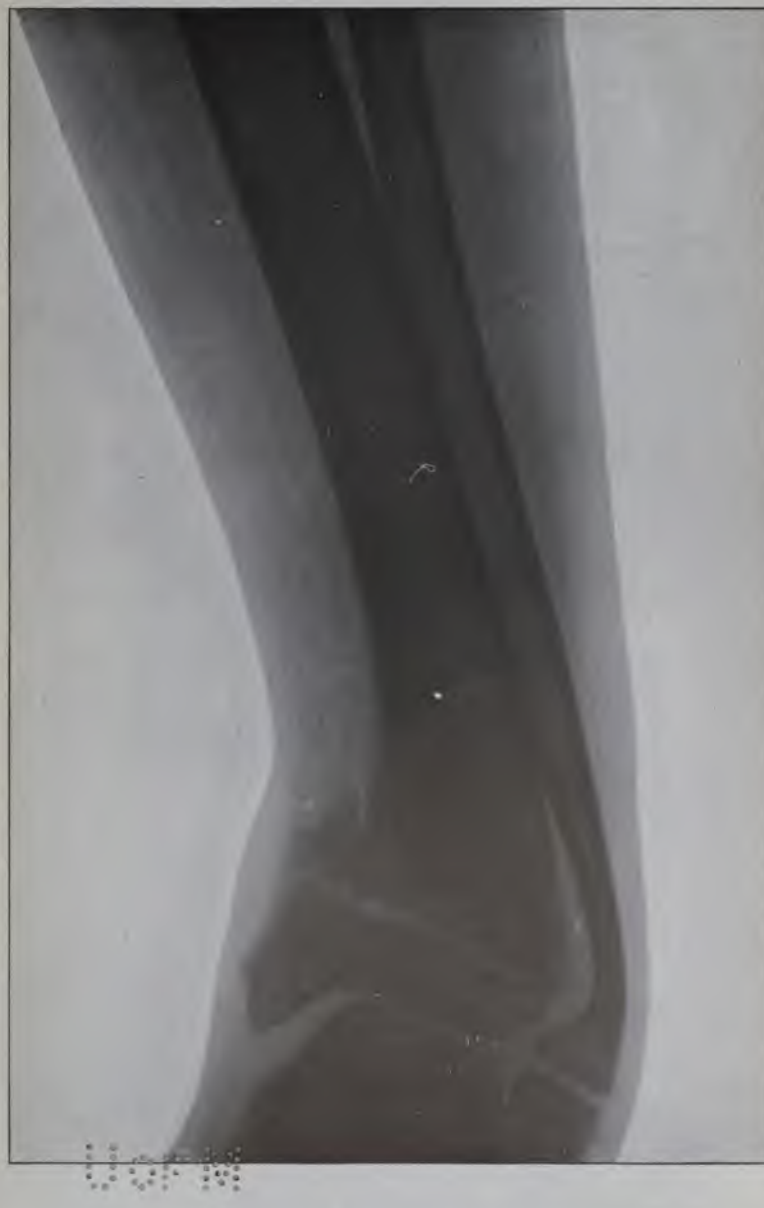

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PLATE III.

Illustrating Mr. Clutton's Cases of Adolescent Rickets.

Radiograph showing the ankle in Case 2.

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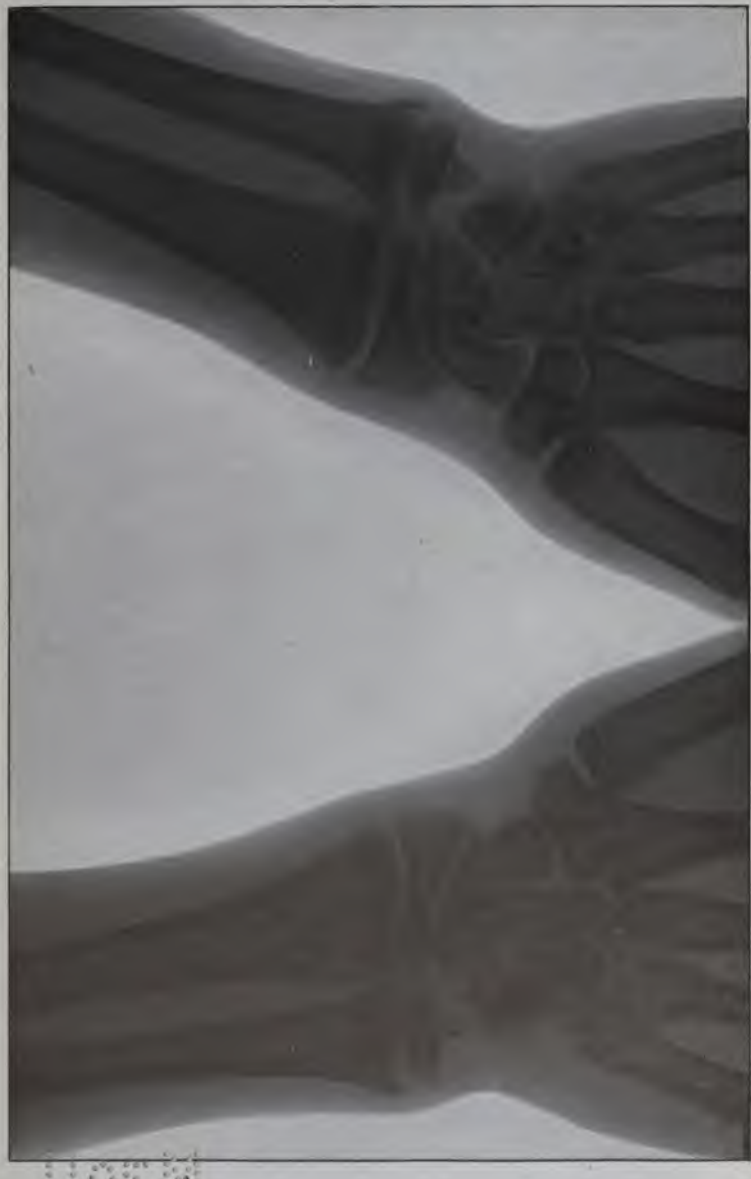

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PLATE IV.

Illustrating Mr. Clutton's Cases of Adolescent Rickets.

Radiograph of the wrists in Case 3.

place between the epiphysis and the shaft. For there is still a wide gap at the epiphysial line in some of these bones, even in those in which there is no proliferation of unossified bone, where there should be only a faint line at the age of twenty years.

In Case 2 osteotomy of the left femur was done in October, 1906. This was followed by imperfect union, and she left the hospital in a steel support. She returned in June, 1907, having sustained fracture of the right femur from a fall. It was then found that there was still only fibrous union of the left femur.

Case 3.—This case was kindly reported to me by Mr. C. A. R. Nitch, resident assistant surgeon to St. Thomas's Hospital. The patient, æt. 12, was an only child. As an infant she was bottle-fed, as her mother died soon after her birth. There were no signs of rickets in infancy, and her health was good. Genu valgum was first noticed in June, 1905, and she was taken to the hospital a few weeks later. There were no signs of active rickets when she was admitted in July. The epiphyses were not enlarged and there was no albumen in the urine. Double osteotomy was performed by Mr. Nitch in August, and the bones united well. About the middle of July, 1906—*i. e.* one year after operation—the wrists were first noticed to be swollen, and when seen at the end of September by Mr. Nitch there was a definite enlargement of both radius and ulna at each wrist. No other bones were affected, and there was no albuminuria. Dr. Greg by the X rays found changes typical of rickets in both wrists, more marked in the ulna than in the radius. All the other bones examined were normal. If you look at the skiagram (Pl. IV) of the wrists you will, I think, see confirmation of the history that has been given—namely a duration of only a few months. There are a sharp margin and cup-shaped edge at the lower end of the ulna. In the more advanced case last seen the lower edge of the diaphyses has lost its definition. The shaft is, of course, of normal density.

It would be extremely interesting, and important to our patients and their friends, if we could explain how and why this disease arises. I cannot pretend to be able to do this, but I should like to make a few remarks.

Adolescent rickets must, I think, be looked upon as practically the same disease as infantile rickets, modified in

some respects by the age of the patient. The question naturally arises as to whether the adolescent form is merely a continuation of infantile rickets or entirely a fresh and distinct development. It might, of course, be a recrudescence of what the child had suffered in infancy. Against this latter view is the fact that on recovery from infantile rickets the bones are, as a rule, sclerotic, a condition in which one would imagine a relapse would be unlikely to occur. On the other hand, if it be considered to be a continuation of infantile rickets, the first attack must have been very mild in severity and without any tendency to recovery. For there is no history to be obtained of sufficient severity by which we can say definitely that the patient has suffered from rickets in infancy. I am inclined, therefore, to look upon adolescent rickets in many cases as a *distinct* development of the same disease arising in circumstances which are chiefly due to the age of the patient. If this be accepted we must assume that rickets may arise at any age from infancy to adolescence. In a great measure one would imagine the same causes would be at work to produce the same changes in the bones. Although we naturally attribute to diet the main responsibility for infantile rickets, we do not deny the influence of the hygienic surroundings of the patient's home. In fact, whatever interferes with metabolism of the body would be likely to prevent the normal development of the bones. One of the most important of these influences upon metabolism must be the higher nerve-centres which, unimportant in infancy, are quickly developing towards the age of puberty. We must, therefore, I think, acknowledge that new factors arise which are scarcely present in infancy, and which are of as much importance as food, if not more. The period of from twelve to fourteen years of age is a time of great intellectual stress from the educational point of view, and anyone who has seen much of children will know what this may mean to many. In some children the competition of school life may produce all sorts of nerve trouble, from insomnia and night-terrors to epileptiform convulsions or facial distortion and mimicry. The same intellectual strain and competition will continue throughout the adolescent period of life or even longer. This is more important than diet at this age, for if it be a cause of this trouble it is difficult to eliminate, and accounts perhaps for the long continuance of the adolescent cases. It is noticeable that in two of my patients diet can have had but little influence, as they were the children of fairly well-

to-do parents. In the first case there is evidence of prolonged mental strain during school life, and in the second the patient had reached the sixth standard of the board school at from twelve to thirteen years of age. The latter fact, perhaps, only proves that she was not a backward child.

Sexual development is another factor and may have a far-reaching effect. It is difficult to judge of this, but in one of my patients (in a case recorded in 1886) premature sexual development was a marked feature. Want of air and exercise must also have a definite influence when once the disease has started, and may therefore be considered as a contributory cause. It probably has also a primary influence among the poor. I would look, therefore, for a variety of causes to produce such results as are seen in the patient, whose age is now twenty-three years and whose wrists showed signs of active rickets two years ago.

There does not appear to be any special treatment applicable to cases of adolescent rickets. The habits and conduct of the patient must be inquired into and action taken in accordance with what is discovered. Diet, education, and fresh air are likely to be the most important points for inquiry. I am inclined to think that correction by osteotomy of a deformity due to this disease should be postponed till all active signs of rickets have disappeared. We occasionally see a recurrence of deformity after correction by operation. It is possible that this is due to the continuance of the same disease which caused the deformity. In the first patient, now aged twenty-three years, thyroid extract was given for six months last winter. I cannot say for certain that his improvement is due to this drug, but the fact remains that he now feels stronger than he has done for some years, and that his lower limbs are better able to support the weight of his body. The bones of the wrists also show, I think, a marked improvement compared with their condition in 1904.

There are a large number of radiographs on the table besides those which I have shown by the epidiascope. These were all taken by Dr. Greg and presented to the museums of the Royal College of Surgeons of England and St. Thomas's Hospital. I am indebted to Mr. Shattock for the opportunity of showing them here to-night. There are also two specimens on the table. One is from the museum of the Royal College of Surgeons of England. It is a specimen of a rickety femur from a girl æt. 17, and was presented to the museum by Mr. E. Muirhead Little, who has kindly consented to say a

few words on the subject. The other specimen, from the Hospital for sick Children, Great Ormond Street, is a femur from a child æt. 12, who was under the care of Sir Thomas Barlow in 1887. The disease had been noticed from the age of five years. The patient died from tuberculosis.

FIG. 2.

FIG. 1.

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PLATE V.

Illustrating Mr. E. Muirhead Little's Case of Adolescent Rickets.

Radiographs showing the wrists.

COMMUNICATIONS.

I.—*Case of Late Rickets.* By E. MUIRHEAD LITTLE, F.R.C.S., introduced by the PRESIDENT. *Read October* 12, 1906. *With Pl. V.*

THE patient, Minnie R., from whom the specimen to which the President has referred was obtained, was a female, æt. 17. As the case has been described in detail elsewhere (*Medical Press and Circular,* May 23, 1906), I will only briefly mention its main features. She was quite well and able to run about until she was five years of age, when her knees began to give way. She walked about in instruments until she was seven years old, when she took to crutches. Her right femur was fractured by a fall when eight years old and her left one a year later; they united well. When she came to hospital in 1900 she weighed, although not thin, only 49½ lb. and measured recumbent 44 in. Her sexual development was very backward. The urine contained nothing abnormal, but was markedly deficient in calcium salts. She had extreme knock-knee and forward bowing of the femurs. Her ribs were beaded, and her wrists and the lower epiphyses of the femurs were enlarged. These latter were on subsequent osteotomy found to be very soft, and they were so pervious to the X rays that satisfactory radiographs were only obtained with difficulty.

I should like to call attention to the radiographs of the wrists, which are reproduced on Pl. V. They show very wide, ill-defined epiphysial lines and distinct cupping of the ends of the diaphyses. As I pointed out six years ago, this cupping is characteristic of infantile rickets when it affects the ends of the long bones, especially the lower ends of the radius and ulna. These radiographs—of a girl of seventeen—are very much like those of a child of two, the subject of severe rickets. The chief differences are that in the latter the cupping is more marked and the epi-

physial line less blurred. The patient died while in hospital with acute cerebral symptoms. *Post mortem* miliary tubercle was found in the lungs. One femur and one radius were sent to the Royal College of Surgeons. A glance at the specimen now on the table will show the changes characteristic of rickets. The islets of cartilage in the newly formed bone are particularly noticeable and characteristic.

The case is, I think, remarkable for the very long continuance of the disease in an active stage.

The boy whom by the courtesy of the President I am permitted to show to-night offers a far less striking picture than did Minnie R. The following are short notes of the case: William L., æt. 15½, has had no acute disease except pertussis when four years old. Nothing else wrong was noticed until he was eleven years old and attending school, when his right leg began to give way and the knee to yield outwards. He suffered no pain and there is no record of over-strain or injury. From August, 1905, until February, 1906, he wore an instrument but without any improvement of the deformity resulting.

He is a well-nourished, fairly well developed lad, 5 ft. 3¾ in. in height. He has genu varum on the right side. There is a space 3½ in. wide between the knees, which is increased to 5½ in. when he stands up. The head of the right fibula is abnormally prominent. The liver and spleen do not appear to be enlarged. His forehead is rounded and rather prominent. The inner ends of the clavicles and of the lower ribs are enlarged, but the chest is otherwise well formed. The epiphyses at the wrists, knees, and ankles are obviously enlarged. The normal curves of the shafts of the femurs appear to be exaggerated. A radiograph of the right knee shows that the bone is very pervious to X rays and that the deformity is almost wholly due to an outward curve of the tibia in its upper fourth. The epiphysial lines are rather blurred. As will be seen, the bones at the wrist throw an X-ray shadow of almost normal density, but the epiphysial lines are a little wider than normal and the lower ends of the diaphyses of the ulnæ are somewhat expanded and distinctly cupped.

On osteotomy of the tibia and fibula the bones proved to be quite soft. In the absence of a more definite history it is impossible to affirm or deny the previous occurrence of infantile rickets, but it seems to me clear that he now suffers from adolescent rickets.

II.—*A Principle of Treatment suggested for some Untoward Fractures, especially of the Femur.* By E. DIVER, M.D. *Read November* 9, 1906.

IN ordinary treatment of fractures of the thigh the effort of the surgeon is, from the very first, to put the fractured bone in the position it occupied before it was broken, and if he cannot do this, to get as near as he can thereto, then to keep it so for callus to bind it permanently.

The suggested treatment is not like that, but rather, when suitable apposition cannot be secured, to get the fragments merely to *touch* somewhere at their broken surfaces, not making it by any means an essential that the two fragments shall be in the axis of a perfect bone. Indeed, they may be caused to form an angle with each other, and so the contour of the limb be changed for a while, and muscular action sometimes aids to maintain and to strengthen this apparently inimical state of things. For example, say the femur is broken above its middle, the lower fragment drawn to the outer side, and the fracture difficult of accurate permanent adjustment. In treating this, separate the limb sufficiently from its fellow, then so place it that the top of the lower fragment will catch against or under the fractured surface of the upper, or at least shall be near thereto. This position would truly be a deformity for the time being; but muscular action would, in drawing the lower fragment up, make the contact firmer. Maintain this position contact until the broken ends, with consolidating callus about them, are beginning to unite thus crookedly. Then is our opportunity to remove the deformity, and to restore the bone to its natural position; and this is proposed to be done by graduated movement. In proceeding to this, we shall be aided by another natural agency; for the soft-solid callus holding the fragments together will yield to the surgeon's manipulations. Cautiously, therefore, move the limb a little, then give it a due interval of rest. This over, give a further movement, and so on. Each movement should make the apposition more exact and the limb straighter, until in due time it is in symmetry with the other. During each interval of rest the newly acquired position of the fragments will, of course, have to be maintained.

The close similarity of the procedure suggested to that of

a tenotomy in talipes seems to give fair promise of similarly good results. As to appliances, these cannot be discussed in detail, but must be left to the ingenuity and the common-sense of the surgeon, who will suit them to his particular case. But no doubt a good rule and one universally applicable is to dispense with all one can.

In dealing with a fracture, and especially a fractured femur, one's object is, of course, to save the patient in the end from needless bodily infirmity, connoting, as all infirmity does, social disadvantage and coincident mental suffering. The satisfaction which we feel in dismissing a patient merely with a "useful limb," as the permanently damaged member is euphemistically stereotyped, may by one suggestion and another, when approved by painstaking verification, give place to a more satisfactory and honourable expression still. And this, indeed, is my apology for the paper I am reading, which brings a mere suggestion before the Society. It seems to me, however, better that I should make my suggestion rather than await the issue of any case I might treat; for, in my present practice, no fracture of any kind may ever more fall to my lot; and if I may venture to mention another personal matter, I would say that some three and a half years ago I myself suffered from a fractured thigh at the middle third, resulting in considerable shortening, which, partly remedied by artificial means, has left me with the "useful limb" formerly mentioned. A further, and I hope better, issue of my misadventure, is, that thinking over the subject, the suggestion which forms the burden of my paper slowly formed itself. I feel assured I shall have this meeting with me when I say that I shall feel the deepest interest, and much gratitude, to learn the evidence which careful proof of the suggestion in actual fracture may give, as to its usefulness in dealing with poor folk having broken thighs.

III.—*Multiple Stricture of the Ileum. Resection; recovery.* By C. J. Symonds, M.S. *Read November 9, 1906. With Pl. VI.*

GEORGE W., æt. 43, was seen in consultation with Dr. Whitelaw, of Stoke Newington, August 30, 1899.

He stated that since childhood he had been subject to abdominal pain. He married at twenty and his wife, a most intelligent woman, says he began by having one or two attacks of pain a year. These usually came on at night. The pain was of a "tight" character and was accompanied with coiling of the bowel and much noise. He was relieved by hot fomentations and was able to go to his work next day. For the last three years these attacks had become more frequent and for the last three months he had scarcely been free from pain, and in addition had been sick. During this period he had lost twenty pounds in weight. He went away at the beginning of August for fifteen days. For the first six he was fairly well but during the rest of the time was frequently sick, ejecting chiefly a brown-coloured fluid.

On August 30 he looked ill and emaciated and yet possessed a remarkable vitality. There was none of the anæmia that accompanies diffused malignant disease nor was there the lack of energy. The abdomen was big, and on being handled large coils of dilated small intestine rose up and could be seen moving through the very thin abdominal wall. The peristaltic wave was accompanied with loud gurgling and ended with a peculiar sound at a point below and to the left of the umbilicus. This was a long rushing sound as of fluid passing through a small orifice. After the attack was over the thickened coils could still be felt, nor was the abdomen much reduced in size. The outline of the liver could not be ascertained, and there was no reason to think it enlarged. The motions were fluid and fairly frequent. At times the intestine seemed to empty itself and there was relief for some hours. The rectum was empty, nor could any swelling be felt in the pelvis.

The number and situation of the coils indicated the small intestine as the seat of obstruction. Moreover the vomiting and frequent action of the bowels, with the absence of toxæmia, were opposed to obstruction in the large intestine—at least, to any part below the hepatic flexure, and from my

own experience I would say to any part below the ileo-cæcal valve. The obstruction was of the chronic variety and as yet there was no general infection from œdema of the walls.

The rapid wasting was considered to be due to the sickness, to the loss of sleep, and the general lack of nourishment consequent on these conditions, and not to the cachexia of malignant disease.

The two largest coils occupied the ileo-inguinal regions, and just above the larger and left-hand one and below the umbilicus the sound of fluid trickling through an orifice was heard. Other coils were seen between the ensiform cartilage and the umbilicus.

The patient was admitted into Guy's Hospital under my care on August 31, 1899.

On September 3 the abdomen was opened above the umbilicus with a view first of ascertaining the exact nature and position of the obstruction. I thought it might be necessary, on account of the septic nature of the vomited materials, to drain the intestine and subsequently to anastomose or resect. He had been ordered enemata the day before, and the intestine was so well emptied that it was possible to proceed at once to the complete operation.

Besides the dilated coils of small intestine, a large sac, looking very much in size and shape like a thickened stomach, was found in the course of the ileum. At either end a long narrow constriction separated it from dilated and thickened intestine above and from normal intestine below. The bowel being practically empty, resection was at once performed. The bowel was clamped with Doyen's broad ligament clamps, without rubber covering. The mesentery was divided close to the margin of the bowel, after ligation with fine silk ligatures. The thickened and dilated bowel on the proximal side was divided almost transversely, being left a little longer, as usual, on the mesenteric side. The distal portion was cut more obliquely, so as to fit the longer proximal end, but the latter had to be still further reduced by a gusset or tuck. Then the ends were joined by two continuous silk sutures, the first passing through all the coats, the second the ordinary Lembert. Each alternate suture was knotted to prevent pursing. A few additional sutures were inserted where necessary. The omentum was folded over the junction and fixed by sutures. The operation was well borne and no symptoms causing anxiety arose later. The man rapidly gained in weight; his digestion, always difficult, improved at once, so

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PLATE VI.

Illustrating Mr. C. J. Symonds' Case of Multiple Stricture of the Ileum.

Photograph of the portion of ileum excised.

that he has been able to take the usual food. As stated above, he had since childhood been obliged to avoid many articles of diet, such as raw fruit and potatoes, and had taken small and dry meals. Within a few weeks of operation he was able to indulge freely in all kinds of food. At the present time (November, 1906) he is in good health and in full work.

Remarks.

The chief interest lies in the condition of the parts found at the operation, and mainly on this account I bring the case before the Society.

The portion removed was about 15 in. in length and consisted of a thick-walled sac, measuring about 9 in. in length by 3 in. wide, at the point of greatest breadth. At either end was a long stricture of the bowel, evidently of old standing and simple in nature. There were thus two chief points of stricture, with an intervening dilated length of bowel. It is probable that the part between the strictures had also been affected by the same disease, that ended in contraction, and that it was not a simple dilatation. Probably the distal stricture was smaller at first than the proximal, and thus determined the dilatation of the part between.

As to the nature of these strictures, it is probable that they are the result of tuberculous ulceration. The museum at Guy's Hospital contains several specimens of stricture of the ileum, considered to be tuberculous. The symptoms beginning in early life supports this view, though there is no history of any particular illness. In elucidation of the cause I may refer to the case of a child æt 6, admitted under my colleague, Dr. Frederick Taylor, with pain and a swelling in the situation of the cæcum. On handling the part a peculiar smooth grating could be felt, accompanied by gurgling; coiling bowel could also be felt and seen. We attributed the obstruction in this case to adhesions, the result of former appendicitis, as there was a history of several attacks of pain in this region. The ileum close to the ileo-cæcal valve was found thickened and narrowed and in the dilated bowel above were several movable hard bodies. On opening the bowel above the strictures nine cherry-stones, many of them black with age, were removed. These stones had given rise to the peculiar rubbing sound named above. A few days later the

child died of acute peritonitis, the result of the perforation of an ulcer high up in the ileum. There were other ulcers in the ileum and cæcum, all evidently tuberculous. The operation wound in the bowel was secure. The stricture was of old standing, the wall hypertrophied, and the mucous membrane thickened and altered for some distance. It was fortunate, as it turned out, that the decision to postpone resection in the child was adopted. We considered that sufficient relief would be afforded by removing the cherry-stones.

I have met with a second case almost identical with that of Geo. W. The man, æt. 46, was emaciated and very ill indeed. He had been vomiting constantly, and was thought to have pyloric obstruction. On examination, however, the signs pointed to obstruction lower down in the intestine, for the coiling and the sound of fluids passing through a stricture occurred below the umbilicus. This long rippling sound seems to me almost characteristic of stricture of the small bowel. It is higher in pitch and more prolonged than that heard in stricture of the large intestine and quite different from the short gurgle heard at the pylorus. On opening the abdomen a condition almost identical with that of Geo. W. was found. The man was at so low an ebb that it was deemed wiser to anastomose the bowel above and below the strictures. This being accomplished, a Paul's tube was tied in above the junction to drain the bowel, as it was dilated and distended with fluid. This proved an unwise procedure, for the opening never closed. Immediate relief followed this operation, so that the man was able to eat freely, but he became exhausted, partly from the continuous loss through the opening, and partly from some localised peritonitis along the bowel. Unfortunately, I did not see this patient after the operation, having to leave for my holiday, otherwise I certainly would have resected the piece containing the artificial opening. It was a mistake, of course, to tie in the glass tube, and it was unnecessary. The intestine in these chronic cases, not being paralysed, easily forces the contents through the junction, and, again, the bowel can be emptied by pressing on the contents after the junction has been effected. I have little doubt that had I been content with the anastomosis this patient—operated upon some two years before the case that forms the text of this communication—would also have survived. His case is of importance, however, as throwing light on the diagnosis of these conditions. The rapid temporary improvement that followed even in this man, who looked as if the

slightest injury must prove fatal, testifies further to the nervous power possessed by these cases. Emaciated as they look, the nervous energy is still sufficient to endure prolonged operations, and as the whole of the work is conducted outside the peritoneum the shock of an hour's operation is very slight. Personally I have learnt never to hurry in dealing with chronic obstruction, whether in the small or large intestine, provided there be no toxæmia.

IV.—*Rotatory Subluxation of the Atlas.* By Edred M. Corner, M.C. *Read November* 9, 1906. *With figure and diagrams.*

In his *Clinical Essays and Lectures* published in 1902, Professor Howard Marsh has included one on the "displacement and injuries of muscles and tendons," * in which the sixth, seventh, eighth, and ninth cases recorded are of special interest in connection with the subject of this paper. They may be called examples of "traumatic wry-neck," and one of them is briefly reproduced to illustrate the condition.

Case 7.—"A boy, æt. 19, while vigorously washing his face and neck, felt a sudden pain in the side of his neck, and found that he could not move his head. When he came to the hospital three hours afterward, he presented the appearance of a person with a wry neck, and any attempt at movement was very painful. He inhaled gas, and his muscles were kneaded where he had previously indicated that he felt pain, and his head was placed in its proper position. When he had regained consciousness all his symptoms had disappeared. . . . It seems highly probable that *a tendon of one of the deep muscles, approaching its insertion, had slipped over the apex of the posterior tubercle of the transverse process* of one of the cervical vertebræ."

The sixth case quoted by Professor Marsh was one of M. Ponteau's, who in commenting on it said he believed that a "*bundle of the splenius muscle had slipped.*" All four examples sustained their injuries by quickly and rather forcibly turning their heads to one side. Two were easily reduced by anæsthesia, one by manipulations, and one refused treatment.

These cases form an excellent clinical introduction to the one about to be related, in which, also, the accident was not a violent one. The sole difference seems to be in the pathological explanation offered.

Case of Rotatory Subluxation of the Atlas, shown October 27, 1906 (*Clin. Soc. Trans.*, vol. xxxix, 1906, pp. 212–213.):

Case 4.—H. E., a boy æt. 13, dived into a pond which was shallower than he expected, and hit his head on the bottom of it. He did not lose consciousness or power. He

* *Clinical Essays and Lectures*, pp. 127–155 (J. and A. Churchill).

had severe pain in the back of his neck and could hardly move it at all. He was seen by a doctor but would not stay in bed after the first three days following the accident. A month later the patient was brought to St. Thomas' Hospital as an out-patient on account of his stiff neck. He was admitted later.

On examination, it was seen that he carried his head a little flexed and turned to the right. He had limitation of all of its movements, particularly that to its left (or uninjured) side. There was pain on any attempt at movement. His chin was depressed towards his chest. Before admission he was unable to lie on his back and slept lying on his abdomen. The transverse process of his atlas could be easily felt on the left side; but on the right the finger sank into a groove and the process was found to have been displaced backwards. He could open his mouth sufficiently to allow an examination of the posterior wall of his pharynx being made. The left transverse process of his atlas could be felt to be displaced forwards; and on the right side the rounded protuberance of the right side of the axis, which had been laid bare by the backward displacement of the right side of the atlas was apparent. There was a healed scalp wound on the upper side of the left parietal region which had been inflicted by the same accident.

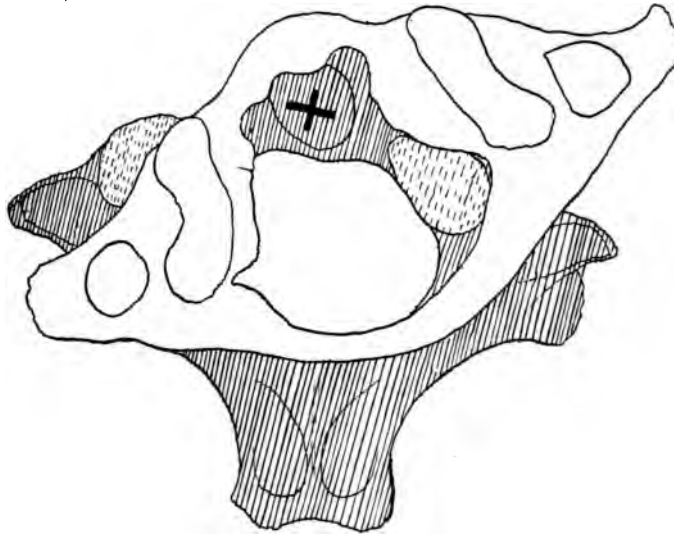
The skiagraph, taken by Mr. Greg, showed the forward displacement of the one side of the atlas.

On the third day after his admission to the hospital chloroform was administered by Mr. Drew, and as muscular relaxation was induced the boy's head slowly rotated to the left and the deformity was reduced without any snap whatsoever. Afterwards the boy was fitted with a collar of poroplastic material, which he wore for a fortnight to three weeks and then left off. When shown to the Society last October he had recovered all his movements.

It will be seen that this case is very similar in kind to those of Professor Marsh and M. Ponteau, though perhaps a little more severe in degree. In explanation of it, it is needful to study some points in the anatomy and physiology of the joints between the lateral masses of the atlas and the body of the axis (see "Physiology of the Lateral Atlanto-axial Joints," *Journal of Anatomy and Physiology*, xli, pp. 149–154). The movements of rotation of the head in a horizontal plane are performed mainly at these joints. When the head is turned to the right, the right transverse

process of the atlas is more or less fixed or is rotated backwards whilst the left moves forwards. If the articular surfaces on the atlas are examined it is seen that they are not plane horizontal surfaces, but from the base of the odontoid process they slope outwards, downwards, and forwards. So that if the right side of the atlas is rotated backwards, it travels uphill, whilst the left side, which comes forwards, travels downhill. The ligaments round this joint have to be lax in order to allow free movement, and our heads in consequence are sustained by unconscious muscular action and not by ligamentous action. If by any mishap this muscular action is overpowered or "caught napping," an injury is par-

FIG. 1.



ticularly prone to result to the side of the atlas which rotates forward and downhill. This injury must be of the nature of a dislocation or subluxation (Fig. 1). The anterior edge of the joint surface of the axis is sharp and clearly defined, whilst the posterior is rounded and obtuse. If this mechanism is examined in partially dissected necks, it is found that the pull of the muscles of the neck locks the atlas on the sharp anterior edge of the facet on the axis. When this is so the head cannot be rotated to the injured side, the one on which the atlas is displaced forwards, whilst it can to the uninjured.

This is very well as a *post-mortem* observation, but can it be applied to the living subject? I believe that it can. If attention is carefully directed to the positions of the trans-

FIG. 2.

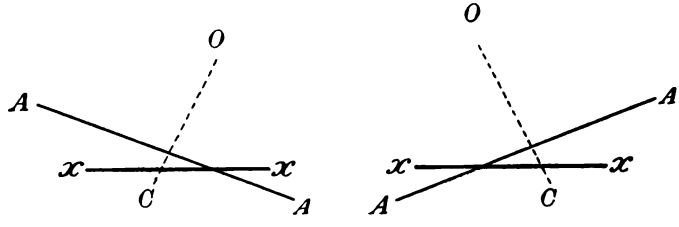


DIAGRAM I.—The normal with "eyes right" and "eyes left."

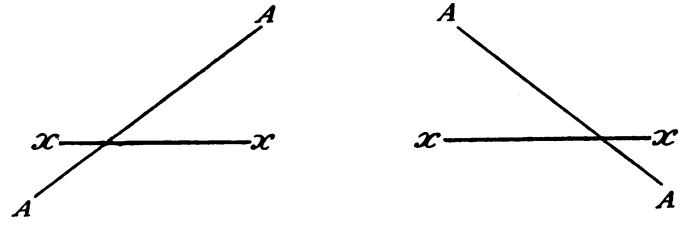


DIAGRAM II.—The normal with the eyes to the left and to the right.

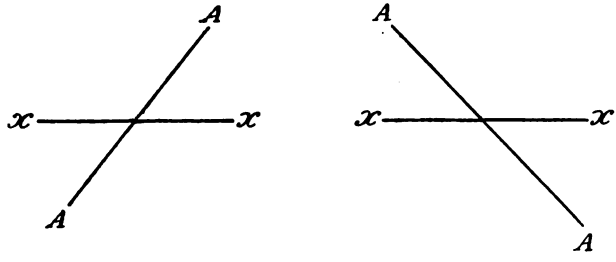


DIAGRAM III.—The abnormal, subluxation of the anterior side of the atlas; the head is not kept to the left but has the eyes turned to the front as much as is possible.

FIG. 2, Diagrams I, II and III.—A A is the long, transverse axis of the atlas, *x x* that of the axis vertebra, C and O are the long axis of the head, C being the chin and O the occiput.

verse processes, the rotation and fixity of the atlas can be demonstrated. In order to make the movements of the head

which are possible in this position clear it is needful to employ diagrams. The long axis of the head is normally at right angles to those of the atlas and axis vertebræ, as when looking straight forward. The transverse axes of the two vertebræ correspond in this position. But if the atlas is rotated on the axis, as in turning the head, and on my hypothesis it becomes locked there, they no longer correspond (Fig. 2, Diagram I).

The displaced atlas carries the head so that it should be moved through a corresponding angle (Diagram I). But as the tendency, as in speaking, is to look straight forward, eyes front, the long axis of the head no longer remains at right angles to that of the atlas, the patient turning his head as much to the middle line as he can—*i. e.* attempting the "eyes front" position. It is from this position that the movements in a case of subluxation have to be studied. From it the head can be moved from the middle line but little or none towards it. The "eyes front" position is assumed in cases of rotatory subluxation of the atlas by means of sliding movement at the occipito-atlantal joints. This was seen in the lateral tilt on the boy's head.

Normally, the transverse processes of the atlas can be felt between the mastoid processes and the angles of the jaw, in the position of Diagram I. If the head is turned as in Diagram II they can still be felt, the movement taking place between the atlas and the axis, not between the atlas and the occiput. But when it is in the position of Diagram III, the angles of the jaw will tend to hide the transverse processes particularly on one side, when a marked groove will be found on the other. This point demonstrates the position of the atlas and confirms the suspicion of its fixation in a position of subluxation. The conditions of the transverse processes are due to the head being turned from the position of Diagram II to that of Diagram III, whilst the atlas is fixed, the angle of the jaw especially obscuring the anteriorly displaced process.

Dr. Schiele (*Deut. med. Woch.*, 1904, p. 100) has recorded four cases of a similar injury. One resulted from the patient sleeping with his head hanging over the edge of the bed—*i. e.* during muscular relaxation. The other three occurred in boys who fell from trees. All were reduced under anæsthesia. He gives some elaborate points of diagnosis, such as the relative heights of the two ears. But as he does not touch on the essential and primary, but rather on the superficial

and secondary, features of the cases, the remarks made are not reproduced. M. Lannelongue (*Compt. Rend. de l'Acad. de Sci.*, Paris, 1904, vol. cxxxix, pp. 495, 496) has recorded two somewhat similar cases which for various reasons have been classed as complete rotatory and unilateral dislocations of the atlas.* The subluxation with which this paper deals is but a half-way house to the unilateral rotatory dislocation of the atlas, an example of which I showed to the Clinical Society on February 24, 1905.† These complete dislocations are more severe than the subluxations, the differences between them being found in the greater degrees of severity —*i. e.* in quantity, not in quality. In the first place, the injury is more severe, the neck more swollen, tender, painful, and stiff, the prominences in the pharynx are more easily felt, the transverse processes of the atlas are more obviously displaced, great difficulty may be experienced in opening the mouth, and so forth.

With regard to the treatment of these cases an attempt should always be made, by means of a good skiagraph and the help of a skilled interpreter of them, to ascertain if the odontoid process or anything else, most likely the anterior arch of the atlas, is broken. Naturally, these additional injuries are much more frequent with complete than with partial dislocation or subluxation. An anæsthetic should be given slowly and in many cases reduction will take place spontaneously with muscular relaxation; gentle manipulations may be required but strong force should never be used. As soon as the head is reduced it should be fixed with sand-bags and the patient carefully watched whilst he is recovering from the anæsthesia. The greater care must be exercised the greater the doubt of the existence of complicating injuries.

Little is known of cases of rotatory subluxation of the atlas, but this much may be anticipated. It will be very infrequently complicated by other injuries, fracture of the odontoid process being the most likely. Because any fracture of the axis or the atlas may be compared to the breakage of a phlange in a key which will prevent it turning the bolt and locking the door, just as surely will a fracture of the atlas or axis prevent the locking of the bones in subluxation.

The relationship between fractures of the base of the odontoid process and subluxation of the atlas is not under-

* *Annals of Surgery*, January, 1907, pp. 9–26.
† *Transactions*, vol. xxxviii, p. 228.

stood.* But it can be suspected that a temporary subluxation has led to the fracture when found alone and without any apparent displacement of the bones, because the subluxation will have been reduced by any, and perhaps temporary, unconsciousness or by death. It is not a condition which is at all likely to be found after death, because it depends on muscular action to keep the bones locked together.

* *Med.-Chir. Trans.*, 1907.

V.—*A Note on a Case of Congenital Deformity of the Spine, etc.* By J. Jackson Clarke, M.B., F.R.C.S., *Read November* 9, 1906.

CHARLES —, æt. 3½, from birth had held his head bent forward on his chest and the shoulders drawn up nearly to the level of the occiput. He could raise his eyes to the horizontal only by arching the body in extreme lordosis. His face presented an appearance as of vertical compression, such as we see in cases of anencephaly, and the whole attitude was reminiscent of that extreme error of development. The boy dribbled, although he had been operated upon for "tongue-tie." He had absolutely no power of rotation or of either lateral flexion or extension of the cervical spine. He had been treated with an antero-posterior splint and massage for one year with very little improvement. I suggested that it would be worth while to put the child under an anesthetic and see if by dividing muscles, etc., any improvement could be obtained.

This operation I did on May 10, 1905. Previous to making an incision I could not decide whether sterno-mastoid muscles were present or not, the neck being so short and the muscles devoid of any prominence. I was pleased to find them and I divided each one on a director. This was done as close to the lower end of the exceedingly short muscles as I could expose; they were wholly muscular—*i.e.* not tendinous—where the section was made. The trapezius I also divided through an incision on each side, above the position of their nerves.

I was now able to extend the cervical region with very little resistance and also to rotate the head from side to side. On completing these movements the patient became very faint. I returned the head to the flexed position and, holding it so, had the boy inverted. The pulse returned but was very rapid, hardly countable, and he was put to bed with the head held bent forwards by the antero-posterior splint. For a week the pulse remained very rapid, but it gradually slowed down to 120 on the seventh day after operation. If I should again encounter a similar case, I shall divide the muscle as in this instance, but I shall extend the neck only gradually after the operation. The extension of the neck on the instrument was gradually increased and on June 22

exercises were very cautiously commenced. The following interesting phenomena were observed in doing them: (1) When the neck was extended patient complained of pain in the epigastrium, and the abdomen suddenly bulged from a sharp contraction of the diaphragm and the pulse increased from 108 (normal for the boy's age) to 122. When the boy's head was turned to the left he had an attack of breathlessness.

By June 28 these phenomena, which evidently depended on the stretching of the nerves in the neck probably acting on the medulla oblongata, were gradually diminishing and by the end of July they had almost ceased and the patient was able to hold up his head without any lordosis, and to turn the head freely from side to side. This improvement is maintained at the present time, and the boy has a neck, if a short one, where he had none before. The right shoulder is still somewhat higher than the left owing to the abnormality in the vertebral column; simple massage and exercises are still continued at the patient's home. His dribbling has ceased.

Skiagraphs.— Study of a series of skiagraphs taken recently show the following points:

(1) The presence of thirteen ribs, a cervical rib being present on both sides of the body. (This feature is only clearly shown in one X ray in which the lower ribs are clearly seen.)

(2) A double lateral curve, to the left in the cervical and to the right in the uppermost dorsal region. This curve accounts for the greater elevation of the right as compared with the left shoulder; thus part of the deformity is the same as that seen in cases of congenital elevation of the shoulder (Sprengel's deformity).

(3) Appearances which indicate defective structure in the lower cervical and upper dorsal vertebræ: (*a*) an unossified space in the front of the column; (*b*) a fusion of the last cervical with the first dorsal vertebra, and (*c*) an increased width in the upper dorsal vertebra, as if the want of ossification referred to under (2) might be due to an anterior spina bifida.

It may be added that the boy is in vigorous health, and seems to be normal in intelligence, although he is backward in speaking, being at the age of four years unable to say more than a dozen words, though he appears to understand perfectly what is said to him.

Only one other similar case has come to my notice, that of an adult lady; no treatment had been adopted and the fixity of the head is complete. In this instance the intelligence and character are both above the average.

I have not seen any previous record of the results of operative treatment in such cases.

VI.—*A Case of Hæmorrhage into the Prefrontal Lobes of the Brain.* By J. Walter Carr, M.D. *Read November* 23, 1906.

THIS case is brought forward because it affords an example of the difficulties in diagnosis which are likely to arise in the somewhat rare instances of hæmorrhage into the prefrontal lobes.

George M., æt. 53, a painter, was admitted to the Royal Free Hospital in a semi-comatose condition about 6 p.m. on June 27, 1906, with a history that about 11 a.m. he had fallen down unconscious whilst at work, had passed urine unconsciously, and vomited some brandy-and-water which had been given him. The man's fellow-workmen at first attached remarkably little importance to the attack and only after he had remained unconscious for several hours did they bring him to the hospital.

A medical man who had attended the patient five years before stated that he then had albuminuria, and since that time he was said to have complained frequently of severe headache and giddiness. For six weeks before the final attack he had been noticed to be getting much thinner, with loss of appetite and frequent vomiting.

On admission the man was almost unconscious and very restless; his temperature was 98·6° F., pulse 56 and of very high tension, pupils equal and of medium size, no squint or conjugate deviation of the eyes; knee-jerks equal and of normal strength. Examination of the chest and abdomen showed nothing abnormal; there was no indication of any cardiac hypertrophy. Ophthalmoscopic examination *nil*. Urine, sp. gr. 1028, almost solid with albumen, gave both Fehling's and the phenyl-hydrazin tests for sugar, but not the reactions for diacetic acid and acetone.

For two or three days the patient remained semi-conscious and restless, but gradually became quieter and able to converse quite rationally with his relations. On July 4, however, he again became restless, then drowsy, and finally passed into a condition of absolute coma, in which he died on July 6, nine days after the first seizure. The temperature was subnormal for the first four days, and afterwards rose gradually but irregularly, varying between 99° and 103° F., with a final rapid rise to 106° F. shortly before death.

After the first day or two the urine no longer gave the reactions for sugar and the albumen also disappeared, save for an occasional trace; the sp. gr. was about 1020; a few hyaline and granular casts were found. It was not possible to ascertain the total quantity passed, but almost certainly it was not above the normal.

There was never the slightest sign of hemiplegia or of any paralysis whatsoever. On July 3 the knee-jerks were noted to be absent.

Post mortem.—A saccular aneurysm, the size of a small pea, was found on the anterior communicating artery at the base of the brain; it had ruptured into the left frontal lobe, the substance of which was extensively destroyed and occupied by a large hæmorrhage. Near the anterior end of the lobe the blood was found to have again burst through the mesial surface and to have entered the right frontal lobe, destroying an area that had a cross-section rather larger than that of a shilling. The hæmorrhage had closely encroached upon the left lateral ventricle, but had not actually ruptured into it. There was considerable thickening of the pia mater around the aneurysm and also between the frontal lobes, with fairly firm adhesions between their mesial surfaces. The vessels at the base of the brain were atheromatous.

The kidneys weighed 4 oz. each. The capsule of the right stripped readily, that of the left was slightly adherent. In both the cortex was somewhat thinned and the consistence increased. Under the microscope an early stage of chronic interstitial nephritis was seen to be present. The heart weighed 13 oz. and did not show any marked pathological changes.

The course of events had probably been as follows: An aneurysm had formed on the anterior communicating artery, as a result of atheromatous degeneration. It was so closely enveloped by thickened and adherent pia-arachnoid that when it ruptured the path of least resistance for the escaping blood was into the substance of the left frontal lobe. A comparatively small hæmorrhage doubtless occurred at first, giving rise to the sudden onset of apoplectic phenomena. The initial fall of temperature usually associated with cerebral hæmorrhage had, of course, passed off by the time the patient was brought to the hospital. The bleeding ceased temporarily and consciousness was regained. About the seventh day, however, the hæmorrhage probably recurred, extended

to the right frontal lobe, and continued slowly until profound coma, hyperpyrexia, and finally death resulted.

The special interest of the case lies in the question of diagnosis. Hæmorrhage into the prefontal lobes, apart from traumatism, is admittedly uncommon, less frequent probably than into any other considerable area of the brain-substance, except perhaps the cerebellum, and when it occurs is not likely to give rise to any localising phenomena. That tumours and also abscesses in this region may not cause any special or local symptoms has been proved repeatedly, but the number of recorded cases of hæmorrhage is much fewer; of 73 cases of lesion of the frontal lobes, unilateral and bilateral, collected from various sources by Dr. Ferrier, four only were due to hæmorrhage (Allbutt's *System of Medicine*, vol. vii). It is true that disease of this part of the brain is especially associated with mental disturbance, but in the case of a hæmorrhage at all large the resultant apoplectic phenomena must necessarily mask, at least for some days, any mental symptoms attributable to the special seat of the lesion. Moreover clinical experience shows that even considerable damage, if limited to one prefrontal lobe, does not necessarily give rise to any marked alterations in character, conduct, or self-control. For instance, Dr. Hale White records in the *Transactions*, vol. xviii, the case of a woman, æt. 31, who two months before her death was seized with epileptiform convulsions, became unconscious and afterwards vomited severely. She completely regained her former condition, and had no motor, sensory, or intellectual symptoms, dying finally from old-standing heart disease. A cerebral hæmorrhage was not even suspected, yet at the *post mortem* a clot about the size of a filbert-nut was found in the white matter of the right frontal lobe.

In this connection it is of interest to note that the present patient recovered his mental power sufficiently to be able to arrange business matters with his wife a few days before his death. At that time the hæmorrhage was almost certainly limited to the right frontal lobe; and it may well be, as is suggested by Dr. Hale White's case, that at any rate the more marked disorders of character and conduct described as due to lesions of the prefrontal lobes, and not resulting merely from a general increase of intra-cranial pressure, occur only when both lobes are more or less damaged.

In the vast majority of cases of cerebral hæmorrhage some paralytic symptoms, usually more or less unilateral in dis-

tribution, are present from the first; consequently when a patient is seen who has suddenly become unconscious, and no such indications are found after careful search, the natural and usually also the correct rule is to try to discover some other cause than hæmorrhage for the condition. The question of diagnosis is further complicated by the fact that, as in this instance, a patient who has cerebral hæmorrhage is frequently suffering also from old-standing kidney disease. Hence it almost inevitably follows that if a man with definite indications of such chronic renal disease becomes unconscious, as a result of a cerebral lesion which does not give rise to any localising symptoms, his condition is likely to be attributed to acute uræmia. Thus in the present case a provisional diagnosis of uræmic coma was made at first, a diagnosis based on the history of albuminuria five years before, followed by wasting, headache, vomiting, etc., for some time before the final attack, symptoms all pointing to kidney mischief, to which the man's occupation as a painter would predispose him. The presence of a large quantity of albumen in the urine when the patient was first seen, the markedly high-tension pulse, and the entire absence of any localising indications of a cerebral lesion seemed to confirm the diagnosis. The question of diabetic coma had also to be considered, but although sugar was abundantly present in the urine at first, yet the patient's aspect was not that of diabetic coma, which was also rendered very improbable by his age and by the absence of dyspnœa or of any special smell of the breath, and of the reactions for acetone and diacetic acid in the urine.

It subsequently transpired that both the albuminuria and the glycosuria were temporary symptoms due merely to the disturbance caused by the cerebral lesion, although the *post-mortem* examination proved the existence of some chronic kidney disease. The chief argument at first against the diagnosis of uræmic coma was the suddenness of the onset of cerebral symptoms, the coma of contracted granular kidney being nearly always gradual in development. Subsequently the almost complete disappearance of the albumen, the period of improvement, and then the gradual rise of temperature, with deepening coma, rendered the diagnosis of uræmia a very improbable one, especially as in cases of uræmic coma the temperature usually remains sub-normal.

On reviewing the whole case it would seem that there were hardly sufficient data for an accurate or tolerably certain diagnosis. It is true that the sudden onset of unconsciousness, the

partial recovery, the temporary albuminuria and glycosuria, and the final symptoms of an ingravescent apoplexy, were probably sufficient to justify a diagnosis of cerebral hæmorrhage; but in the absence of all localising phenomena it was impossible to suggest the exact situation of the lesion, except that it was presumably in one of the so-called "silent" areas of the brain; and the comparative rarity of hæmorrhage in these areas must naturally make the diagnosis uncertain, if the conditions present are such as may fairly be attributed to some more common morbid condition—such, for instance, as uræmia. The restlessness of the patient was very marked during the first few days, but whether this was more noticeable than after similar damage to other parts of the brain is doubtful.

A practical deduction of some importance is that if a comparatively small hæmorrhage should occur into one prefrontal lobe and recovery follow, there may be nothing to indicate that the patient has had so serious a lesion. Dr. Hale White's case, already referred to, affords an example of this.

Fortunately, the main lines of treatment are much the same whether the case be diagnosed as one of uræmia or as one of cerebral hæmorrhage, viz. to lower the arterial tension as much as possible, for this is what all the eliminative treatment for uræmia tends to do. Hence the remedies employed in the present case—purgatives, especially mercurials, pilocarpine and nitro-glycerine—although used at first under the impression that the patient was probably suffering from uræmia, were almost equally suitable for arresting the hæmorrhage which was actually present. Even a prompt venesection, probably the best treatment in the early stage of cerebral hæmorrhage, may do much good, and certainly can do no harm, in uræmia, although if the condition be secondary to contracted granular kidney improvement can hardly be looked for.

VII.—*Cystic Dilatation of the Lower End of the Ureter containing Calculi, which became engaged intermittently in the Urethra.* By W. G. Spencer, M.S.

Read November 23, 1906. *With Pl. VII.*

THE surgeons of the eighteenth century in their observations on the encysted calculi which they met with in the course of lithotomy operations, noted the prolapse of the lower end of the ureter containing a calculus. Houstet in his "Observations sur les Pierres enkistées et adherente à la vessie," in the *Mémoires de l'Académie de Chirurgie*, 1743, vol. i, Part II, p. 268, states that Couillard showed that stones travelling down the ureter to the bladder, instead of passing through the orifice, might insert themselves between the membranes of the bladder-wall. Having recognised by sounding that a stone was encysted, he determined to proceed nevertheless to lay open the cyst, and thus he successfully removed one stone the size of a hen's egg and other smaller ones. Littre in dissecting a young man found two stones contained between the membranes of the wall of the bladder. The stones had penetrated the wall of the ureter in its passage through the bladder-wall, and after becoming arrested between its layers had enlarged. Hence the tearing open of the cyst wall was done by some; other surgeons succeeded in passing a bistoury along the finger with which to divide the cyst wall. Desault, finding a pedunculated cyst containing a calculus, slipped over it his form of uvula-guillotine and cut it off between the bladder wall and the stone which the cyst contained.

Of late years prolapse of the orifices of the ureters into the bladder has been often noticed. Sir Thomas Smith (*Path. Soc. Trans.*, 1863, vol. xiv, p. 185) described a bilateral prolapse of the orifices of the ureters, his specimen being now in the St. Bartholomew's Hospital Museum.

By means of the cystoscope it has been noted that a stenosis of the ureteral orifices occurs, and this is shown by the urine intermittently ballooning the mucous membrane, or a persistent cystic swelling occurs such as was demonstrated to the Society last Session by Dr. David Newman (see *Clin. Soc. Trans.*, 1906, vol xxxix, p. 1, fig. 14). Mr. E. Hurry Fenwick, in his *Handbook of Clinical Electric Light Cysto-*

scopy, 1904, Pl. XV, depicts a case which was excised by suprapubic cystotomy. This operation had previously been done by Mr. Freyer. In the *Med.-Chir. Trans.*, 1898, vol. lxxxi, p. 41, he described an "Obscure Cystic Tumour of the Bladder containing two Calculi" successfully removed by suprapubic cystotomy. In a considerable number of cases the prolapsed end of the ureter has protruded through the urethra of little girls, and four out of five cases proved fatal. Three other papers containing numerous references to cases are:

Blumer.—"Notes on Two Cases of Ureteral Abnormality," *Johns Hopkins Hospital Bulletin*, 1896, vol. vii, p. 174.

Adrian.—"Ein Neuer Operative behandelter Fall von intermittirender cystischer Dilatation des vesicalen Ureterendes," *Archiv f. klin. Chir.*, 1906, Bd. 78, S. 588.

Cohn.—"Cystenartige Erweiterung des Harnleiters innerhalb des Harnblase," *Beitr. z. klin. Chir.*, 1904, Bd. 41, s. 45.

The case I now bring forward is peculiar in the fact that no symptoms were caused, except when the cyst engaged in the commencement of the urethra. There was an absence throughout of any signs of obstruction to the ureter.

A man, æt. 25, had during six years applied for relief to doctors and hospitals. At one hospital he was either sounded or cystoscoped, after which he was told that the left side of the prostate seemed enlarged. But on the whole the symptoms were so vague and intermittent that nothing was proposed for him. He then came under Dr. Davison, of Malden, who, seeing the man in one of his attacks, assured himself that the patient's complaint was real, and on this I admitted him to hospital.

The man's history was that for six years he had intermittently suffered from a burning sensation during and at the end of micturition; or micturition would be much prolonged, he taking ten minutes to empty the bladder. Sometimes he had frequency of micturition by day. Several times in a month he had incontinence of urine at night, waking up to find that he had emptied his bladder during sleep. All this trouble was occasional, not regular; he thought it especially attacked him on cold, wet days. He never had hæmaturia nor passed gravel, nor never acute calculous pains, never any symptoms referable to the kidney or ureter. He had never had gonorrhœa. He was married, was the father of three children, and had no trouble in that connection. During a

Non U

FIG. 1.

FIG. 2.

Bale and Danielsson, Ltd.

PLATE VII.

Illustrating Mr. W. G. Spencer's Paper on Cystic Dilatation of the Lower End of the Ureter containing Calculi, which became engaged intermittently in the Urethra.

FIG. 1.—Photograph of specimen removed from Mr. Spencer's case.

FIG. 2.—Photograph of Sir William MacCormac's specimen in the Royal College of Surgeons Museum.

week he was kept in bed without anything abnormal being discovered; he was thereupon anæsthetised and examined bimanually *per rectum*, when a hard mass could be felt slipping about in the bladder to the left and above the prostate. A sound was then passed and a hard mass struck several times; it did not ring like a stone nor was it fixed in position. As the sound was withdrawn the mass was forced down and became engaged in the prostatic urethra, as if it were a calculus impacted.

On perineal section there was exposed in the prostatic urethra the pedunculated swelling now tightly grasped by the neck of the bladder. The peduncle was traced up distinct from the prostate to the position of the left ureteral meatus. This was drawn down a little and cut through with the actual cautery, immediately above the highest stone, where it was of the size of a lead pencil. The perineal wound healed and the man went home three weeks after the operation. He has reported recently that he has no urinary trouble whatever.

The specimen removed was sent to the Museum of the Royal College of Surgeons (Pl. VII, fig. 1). It is now exhibited and the description of it by Mr. Shattock is as follows:

"3671 A. A somewhat pyriform sacculus about an inch and a quarter in length, and, where broadest, five eighths of an inch in diameter, which projected into the bladder from the neighbourhood of the left ureter. It contains two calculi with crystalline surfaces; a third, which lay above those shown, and which with them quite filled the cavity, was removed for chemical examination. The neck of the sacculus is charred from its having been divided by means of the cautery. Both the interior and the exterior of the sac are covered with a smooth membrane, but close examination revealed no aperture corresponding with that of the ureter in any part of the wall.

"Microscopic examination of the central portion of the sacculus shows it to consist of connective tissue, in the more central part of which lie bundles of unstriped muscle-fibre. On the vesical aspect the connective tissue is invested with a somewhat thick epithelium, of which the deepest cells (when the covering is thickest) are in places set vertically to the subjacent surface; these are succeeded by polymorphou elements, and these again by flat cells in many layers. On the opposite aspect the connective tissue forming the surface is devoid of epithelium; the number of cells in it is

abnormally large, and the surface itself is covered with a thin layer of clot or exudation. The absence of epithelium is, perhaps, to be explained by the inflammatory process set up by the presence of the calculi.

"The sacculus possibly represents a portion of one side of the ureter which has prolapsed into the bladder after the impaction of the calculi in it, the ureteral orifice having probably lain near the base of the protrusion. The calculus examined consisted of phosphate and oxalate of lime; no ammonium nor magnesium was present and no uric acid."

Pl. VII, fig. 2, is the photograph of a specimen in the Royal College of Surgeons Museum, presented by Sir William MacCormac, showing bilateral prolapse of the end of the ureter.

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PLATE VIII.

Illustrating Mr. H. H. Clutton's Case of Ureteral Calculus Causing Retention.

Radiograph showing stone in the ureter at its junction with the bladder.

VIII.—*Case of Ureteral Calculus Causing Retention.*

By H. H. Clutton, M.C. *Read November* 23, 1906. *With Pl. VIII.*

THE interest of this case lies in the fact that it is supplementary to Mr. Walter Spencer's case just recorded. Retention and pain were the prominent symptoms in both cases, and led to a bimanual examination of the bladder by which a diagnosis could be made. By the kindness of Dr. Muzio Williams I was asked to see a youth æt. 20 on September 19, 1906. His history was as follows :

In the preceding July without any premonitory symptoms whilst in the act of passing water the stream would suddenly stop and the patient would have to wait before being able to resume the act of micturition. This occurred several times and was followed by great pain in the right side of pelvis and the passage of blood.

On August 13 he had complete retention of urine, for which a catheter was passed and 30 oz. of urine withdrawn. On September 7, when he came under Dr. Williams' care, he had another attack of retention, which was relieved after several hours by the passage of a calculus.

The intermittent and frequent attacks of pain, which were gradually increasing in severity, were situated in the right iliac region and passed like a wave over the bladder to the base of the penis. The right kidney was free from pain or tenderness. He had been given morphia before he came to London for its relief. This was not continued by Dr. Williams, and when I saw him on September 19 he had acute tenderness at the brim of the pelvis, where the right ureter is situated. On an attempt at standing the pain was so acute that he had to be assisted in getting back to bed. At the consultation on September 19 an anæsthetic was given and the bladder examined with a sound. No evidence of a stone was obtained by the sound. I therefore examined by the bimanual method with the right forefinger in the rectum and the left hand over the pubes. I could then distinctly feel a very hard oval body in the position of the right ureter where it joins the bladder. This body, which I felt sure from its consistence was a stone, could be moved with tissues in which it was imbedded, but it could not be altered in position.

A radiograph was taken by Dr. Greg and a stone shown to exist in that position (Pl. VIII).

On September 27 the bladder was opened by the suprapubic route. Dr. M. H. Spencer gave the anæsthetic, whilst Dr. Muzio Williams assisted at the operation. The stone, covered by mucous membrane, was felt projecting forward like a tumour into the cavity of the bladder. It might easily have engaged the orifice of the urethra and caused retention. An attempt was made to find a way through the ureteral orifice, but even with Dr. Williams' assistance *per rectum* the opening, which was situated behind and to the outer side of the projecting swelling, could not be found. The mucous membrane was therefore grasped by a pair of forceps and brought into the suprapubic wound. The ureteral orifice was then easily seen and enlarged with the knife and the stone extracted. The calculus was in a pouch beneath the mucous membrane to the inner side of the ureteral orifice. He was entirely relieved from all pain and the wound was completely healed on the twenty-third day.

IX.—*The Mimicry of Malignant Disease in the Large Intestine.* By B. G. A. Moynihan, M.S. *Read December* 14, 1906.

WITHIN the last three years I have operated upon six cases of disease of the large intestine under the mistaken impression that I was dealing with examples of malignant disease. In all, the clinical manifestations and the macroscopic appearances supported my opinion, but minute examination of the specimens removed or the subsequent clinical history of the case has proved that, in all, the condition was of a non-malignant character.

It is well known that tubercular disease, especially when affecting the cæcum or the ascending colon, may produce symptoms and signs which are with difficulty distinguished from those due to cancer. I have twice performed colectomy for supposed carcinoma, removing the cæcum and a part of the ascending colon in one case and the cæcum, ascending colon, and a part of the transverse colon in another, when an examination of the tumour removed displayed the undoubted evidences of tubercular disease. But such cases are not very uncommon, and it is not with them that I propose to deal in this paper.

It is not, however, so well recognised that tumours of the large intestine, of the sigmoid flexure more especially, may present all the characteristic signs and symptoms of malignant disease, and yet prove to be nothing more than simple inflammatory conditions. The six cases whose details are given differ in character very considerably; the one circumstance common to them all is the close resemblance, indeed the identity, of the symptoms and signs with those of malignant disease. The following are the notes:

Case 1.—A. C., æt. 28, admitted into the Leeds General Infirmary on April 2, 1904, complaining of passing blood in the motions and of frequent and irregular action of the bowels.

History.—The present illness commenced three months ago. Her life has been medically uneventful save for pneumonia seven years ago. There was no history and no evidence either of syphilis or of tubercle. Aperients were occasionally necessary, otherwise the bowels were regular in their action until

the present illness. Blood was first noticed in the stools in the early part of January, 1904. It rapidly increased in amount and the bowels acted five or six times a day, each motion containing more or less blood. As much as half a pint of mixed blood and mucus was passed at a time. In consequence she became very weak and had to take to bed. About two months ago she began to vomit frequently, almost all food being rejected. This subsided for a time, but recommenced a fortnight later and persisted irregularly up to admission. The passage of blood meanwhile continued and became rather worse. The bowels acted seven or eight times a day, the stools consisting principally of blood and mucus. Occasionally clots were passed. During the few weeks previous to admission she experienced a good deal of aching pain in the left iliac fossa, which continued to within a short time of her coming into the hospital.

On admission she was obviously much reduced and very weak and bloodless from the continued hæmorrhage. She vomited once or twice on the two days following admission. There was no complaint of pain, but the bowels acted from five to seven times a day, a considerable quantity of blood and some mucus being passed. The abdomen was distended and the intestine obviously obstructed.

On rectal examination the sphincter was lax; a soft, somewhat irregular mass was to be felt involving the whole of the rectal wall. Anteriorly it appeared to protrude somewhat, but elsewhere felt like thick moss growing on the wall of the intestine. Its upper limit could not be reached. The rectum from about two inches above the anus was narrowed to such a degree as to make the passage up of the finger very difficult. There was slight bleeding as a result of the examination.

A diagnosis of a rapidly growing carcinoma, with partial obstruction, was made, and on April 17, 1904, *inguinal colotomy* was performed. After the gut was opened the bowels acted at first very copiously, afterwards from one to three times a day. Some blood-stained discharge continued from the rectum, but this was less in quantity than before. Her general health improved considerably for a time. The discharge, however, began to increase and both blood and clots were passed. There was also considerable pain in the sacral region.

On June 16 she was readmitted after having been at home six weeks. The colotomy was then acting two to three

times a day. With the fæces a little blood-stained mucus was passed. The rectal condition was apparently much the same as on the occasion of the previous examination. The patient was seen by Mr. Ward, under whose care she had been admitted, and resection of the growth was deemed advisable.

Operation.—On June 30, 1904, *proctectomy* was performed. The patient having been anæsthetised in the ordinary dorsal position, the colotomy opening was plugged with gauze. An incision was then made from it downwards and inwards for about three inches. The peritoneum having been divided, the sigmoid was withdrawn and divided between clamps about three-quarters of an inch below the artificial anus. The lower end of the upper portion was then closed with a continuous suture of Pagenstecher thread. The upper end of the lower part of the sigmoid, having been sterilised by the cautery, was wrapped in gauze. The patient was now put up in the Trendelenburg position. Clamps were then applied to the meso-rectum close to the sacrum and this fold was divided with the scissors close to the clamps. This proceeding was repeated lower and lower down until the whole of the upper part of the rectum, together with the meso-rectum and its contained glands, was freed. The peritoneum of the pelvic floor was then snipped through with scissors at the bottom of Douglas's pouch and by means of the finger this opening was enlarged and the rectum down to the levator ani freed. The clamps having been ligatured off, the whole of the gut thus detached was wrapped in gauze and packed into the pelvis. The abdominal wound was then closed.

The patient was next placed in the prone position with the thighs flexed over the end of the table, the knees being supported on a stool. An incision was made in the middle line from the centre of the sacrum to the anus, bifurcating anteriorly to encircle that orifice. The last sacral vertebra and coccyx were removed and the lower part of the rectum dissected free, as in the usual operation of proctectomy. There was very little bleeding in this part of the operation, and the upper gauze-enwrapped part of the gut was soon reached and pulled down. The removal was completed by dissecting away the anal canal and orifice, the lower part of the gut not being opened. The pelvic floor was restored as far as possible with catgut sutures and the wound closed, large gauze drains being inserted anteriorly and along the sacrum. The operation lasted nearly an hour and a half.

The patient stood the operation fairly well. The gauze in the colotomy opening was left in three or four days, but the abdominal wound was infected from it, and some superficial suppuration occurred. It was, however, completely healed within three weeks of the operation, as also was the sacral wound. Recovery was otherwise uneventful.

The patient is now (October, 1906) perfectly well. She has gained weight, so that she is now heavier than she has ever been, and her appearance is that of a healthy, well-nourished woman.

Description of the part removed.—The specimen after hardening in formalin measures 9 in. in length. The mucous membrane is greatly thickened and its surface is raised up into prominent fleshy folds and convolutions. Over a part of the specimen there is a considerable degree of ulceration, which, by a process of undermining, has resulted in the formation of bridges and overhanging flaps and festoons of thickened mucous membrane. The thickness of the walls varies greatly, some of the tuberous projections of mucous membrane having a diameter of nearly half an inch; these projections are separated from one another by branching sulci in such a manner as to give to the surface of the mucous membrane over the upper one third of the specimen an appearance somewhat resembling the cerebral cortex. In the lower part of the specimen ulceration has reduced the thickness of the mucosa in places to 1 or 2 mm. The muscular coat averages about 2 mm. in thickness, while external to this, in the lower part of the colon, is a layer of fat, two thirds of an inch thick. All the outer coats of the bowel are densely thickened and fibrous, and the lumen of the gut here is probably only about one third of the normal. The inner surface of the bowel does not present any definite isolated tumour or polypoid growth, but there appears to be a general thickening of the mucosal rugæ, the tags of mucous membrane in the lower part of the specimen being detached at one extremity by ulceration.

Microscopical examination shows an overgrowth of the mucous glands. The glands are of irregular form and separated from one another by dense accumulations of lymphoid cells. Between the bases of the glands many smaller glands are seen cut in transverse section; these, as a rule, present a very small lumen. Sections through an ulcerated patch show much the same appearances about the bases of the gland where these are not disintegrated. Here

there is more infiltration of the submucosa with round cells.

This condition is one quite unlike anything I have met with before. As far as I am aware no similar manifestations have been previously described, and the disease lacks a name. It is certainly not syphilitic, nor is it tuberculous. Of malignancy there is no suspicion. The only specimen in any degree resembling this that I have been able to discover in a search through the literature is figured by Koch (*Archiv f. klin. Chir.*, Bd. 70, 1903, p. 891).

Case 2.—*Perforative sigmoiditis; localised abscess.* Mrs. H., æt. 58, seen in consultation with Dr. Norman Porritt, Huddersfield. The history was that the patient had always enjoyed exceptionally good health up to six years ago, when after eating some tinned chicken and ham she had ptomaine poisoning and was ill two months. Four years ago the patient had a severe illness, with much pain in the left iliac region. The doctor who attended her at that time told her she had "just escaped peritonitis." In this attack she was confined to bed for six weeks. Since then she had suffered from time to time from attacks of pain in the left iliac region, many of which have kept her confined to bed for a few days.

In November, 1904, what proved to be a severe illness began with acute pain across the abdomen; this was followed by constipation, sickness, and abdominal distension. In spite of treatment by enemata, etc., the condition of obstruction grew worse and on December 4, 1904, I opened the abdomen. There was considerable distension of all the intestines, and peritonitis, with serous effusion. The seat of the trouble was found to be in the sigmoid flexure, a little above the brim of the pelvis. The bowel here for about three inches was thickened, swollen, and very hard. A perforation was found on the upper side, and a very copious deposit of lymph was plastered over and around this. The condition was thought to be one of malignant disease of the sigmoid flexure, with a perforation of a deep ulcer in the growth. A large tube was passed down to the open ulcer and the rest of the abdomen closed. The patient's condition, to my surprise, gradually improved; but on December 20, 1904, a deep-seated mass between the median incision and the left iliac crest was found. The patient's temperature rose and a second operation was performed, some pus being evacuated

from an incision two inches to the left of the former one. The abscess cavity reached to the sigmoid flexure. The pus-collection was clearly the result of insufficient drainage at the time of the original operation. The subsequent progress was good.

Dr. Norman Porritt kindly saw the patient on October 1, 1906, and reported: "The patient did well and is now better than she has been for years. All the discomfort in the left iliac region which was more or less always present after the ptomaine poisoning has entirely left her, and the bowels, which never acted without medicine, are now spontaneously regular."

The condition in this case has become clearer to me since I operated upon one of the cases related below. I think there can be little doubt that there was a perforation of a false diverticulum of the sigmoid flexure.

The appearance and the consistence of the tumour of the sigmoid flexure were such that we felt no doubt that malignant disease was present. The tumour resembled in all particulars that which I removed from Case 5, recorded below.

Case 3.—*Pericolitis transversa; colectomy.* Mrs. W., æt. 50, was seen in consultation with Dr. Carlton Oldfield and Dr. Greenwood in May, 1905. She had suffered for some months from occasional sickness and indigestion. During the few weeks preceding my examination of her she had noticed great irregularity of the bowels. Constipation had been most obstinate; when relieved by an aperient there was diarrhœa for twenty-four or forty-eight hours, followed again by constipation for several days. The patient was a very stout woman, but had lost nearly two stones in six months. On examining her abdomen, a hard mass was felt situated to the left of the umbilicus. This mass was about four inches in diameter, and apparently adherent to the parietes. A diagnosis of a carcinoma of the transverse colon was made, and operation advised.

Operation (May 31, 1905).—On opening the abdomen the peritoneum was found to be indurated and gristly, about half an inch in thickness. Involving the transverse colon and the adjacent omentum was a very hard, flat, irregular mass. The induration infiltrated the bowel and the omentum very widely. No enlarged glands were perceptible. No doubt whatever was felt by any of us that the mass was a

malignant growth in the colon. The whole tumour, with about five inches of the transverse colon, was removed, and an end-to-end anastomosis performed.

When the intestine was laid open it was found to be firmly embraced and not a little constricted by a densely hard "scirrhus" mass. The most careful examination of the mucosa revealed no surface defect; the membrane was everywhere smooth and supple; there was no ulceration and no diverticula could be discovered. Beneath the mucosa, which was freely movable on the deeper structures, was a wide infiltrating stratum of fibrous tissue which involved all the other coats of the intestine, and spread into the omentum.

Microscopically the mass surrounding the gut is seen to consist of dense fibrous tissue, containing here and there small aggregations of lymphocytes, particularly around the vessels. The mucous membrane is normal. The origin of the inflammatory process in this case was not discovered. It would appear to have been an inflammation of the peritoneum surrounding the transverse colon, with an implication of the contiguous omentum. No affection of the appendices epiploicæ was found. A condition of things exactly similar to this is known to occur in the sigmoid flexure. (See *Sigmoidites et Périsigmoidites*, Saillant, Paris, 1906.)

Case 4.—*Growth in rectum; proctectomy.* Mrs. S., æt. 41, seen with Dr. Waddington, Bradford. She gave a history of having suffered from diarrhœa for five months, small quantities of mucus at times tinged with blood being passed, sometimes as often as twenty times in the day. The rectum on examination was felt to be filled with a very soft, friable, ulcerating mass, fairly movable though extensive.

On July 17, 1905, a preliminary inguinal colotomy was performed, and a month later the removal of the growth was undertaken. Eleven hours before the second operation 27 c.c. of a 2 per cent. solution of nucleic acid were injected subcutaneously. At the commencement of the operation, at 9 a.m., the temperature was 100·4° F., pulse 120. The pelvic colon and rectum down to within an inch or two of the anus was removed by the trans-sacral route, the upper portion of the pelvic colon being fixed in the upper angle of the wound. At 2 p.m. the temperature was 100° F., pulse 140; at 10 p.m. temperature 98·6° F., pulse 95.

Macroscopic appearances of specimen.—The specimen measures 14 in. in length and includes portions of the rectum

and pelvic colon. The upper eight inches of the bowel are normal, but below this the mucous membrane is covered with an exuberant cauliflower-like growth. At the junction of the healthy with the affected portion the mucous membrane is undermined; below this is a flattened area which appears to be superficially ulcerated; adherent to it in places are tags of villous growth. The lowest portion of the bowel, extending to within an inch of the bottom of the specimen, is occupied by a soft and friable projecting, papillomatous mass which is extensively undermined, forming a kind of bridge over a portion of the bowel. The whole circumference of the intestine is involved for a length of about four inches. The obstruction to the bowel, partly by the exuberant growth, partly by a thick fibrous deposit in the wall of the intestine, is well marked.

Microscopical report.—Sections of the villous growth show delicate branching processes composed of a framework of fine connective tissue and covered with a single layer of tall columnar cells whose nuclei are larger and stand more irregularly at different levels than those of the cells covering the non-villous mucosa. Sections of the flattened areas of mucous membranes show the bases of the intestinal glands strictly limited by the muscularis mucosæ; but more superficially the cells are desquamating and lie in many places entangled in a mucoid substance around the broken-down gland mouths. The lymphatic glands examined contained no epithelial deposits.

In the majority of the sections there is no evidence of malignancy, but in one place sections show irregularly-massed epithelial cells extending deeply towards, but not invading, the muscular coat. The clinical history, and the conditions found on examination, both justified a diagnosis of malignant disease in this case. Yet the condition is a simple one. There are multiple exuberant papillomatous growths undermined by ulceration, and a considerable thickening of the outer coat of the bowel due to a deposit of fibrous tissue has caused a moderate degree of stenosis.

Case 5.—*False diverticula of sigmoid; sigmoiditis; colectomy.* Mr. A., æt. 52, seen with Dr. Helm, of Carlisle, April 2, 1906. He has suffered from stomach troubles for a long time, but dates his present illness from eighteen months ago. He has attacks of pain in the abdomen which come on at varying times after food, from half an hour to three or four

hours. He also suffers from a sense of weight and distension in the stomach, and frequently vomits. He vomited blood on one occasion twelve months ago. The bowels are habitually constipated. During the last few months this has been a more serious inconvenience than before. On a few occasions, constipation has persisted for three to six days, and has given way only to persistent effort. On two occasions, five weeks ago and ten days ago, there was intestinal obstruction. The abdomen became greatly distended, obstruction was complete, and vomiting of intestinal contents occurred. The last attack was so serious that it was feared operation might have to be undertaken for a possible growth in the large intestine.

On his admission to the Nursing Home the stomach was found to be slightly dilated; no contractions were seen and no tumour felt. The gastric contents on admission to the Nursing Home showed the presence of free HCl and contained sarcinæ. After a twelve hours' fast, the stomach contained some food residue, yeast and sarcinæ, but no HCl or lactic acid. One hour after a test meal HCl was present in excess. There was a moderate digestion, leucocytes one hour after a meal.

Examination *per rectum* revealed nothing. The diagnosis was chronic duodenal ulcer with stenosis. A fear was entertained that there might also be a growth in the large intestine, though nothing could be felt.

At the operation, on April 4, 1906, an inflammatory mass was felt in the duodenum. It was hard and fibrous, of the size of a walnut; there was evident duodenal obstruction. The large intestine was then examined from the transverse colon, which was distended a little downwards. On reaching the ilio-pelvic colon, a hard mass was felt which was thought to be malignant. As this was deemed the more important condition, the duodenal ulcer was left untreated and a fresh incision made over the tumour, which was very adherent to neighbouring coils of small intestine and to the abdominal wall. The adhesions were separated and the tumour delivered into the wound. The affected part of the sigmoid flexure, in length about five inches, was excised and axial anastomosis performed. The patient recovered, convalescence being somewhat delayed by infection of the upper wound which was sutured last. On October 14, 1906, the patient reported that he had "made very great progress." The following is the report upon the specimen:

"*Macroscopical appearances.*—The portion of bowel removed

is about five inches long; the walls are greatly thickened, making the unopened specimen appear to be involved in a growth. The external surface of the gut is reddened, lumpy, and in places flakes of lymph mark the site of recent adhesions to neighbouring coils of small intestine. On laying open the bowel, the mucosa shows no sign of growth or ulceration, though its lumen is slightly contracted by reason of the induration of the gut. This, in certain parts, is over one and a half inches in thickness, the increase being due to inflammatory exudation beneath the peritoneum. When the mucous surface is examined more closely, numerous pockets or diverticula are seen, some of which might contain the tip of a No. 10 English catheter. On cutting sections of the bowel wall parallel with the long axis of the pockets these are found to be of variable depth, often twisted or slightly dilated at their blind extremities. The pouches are lined by thinned mucous membrane and have pushed the circular muscle layer before them. Around the tips of the deeper pockets the muscular coat has become atrophied, but is traceable as a well-defined, white, fibrous-looking layer continuous with the unaltered muscular coat elsewhere. The extremities of one or two pouches are surrounded by an area of inflammatory extravasation, though no perforation can be actually demonstrated.

"*Microscopically.*—The pockets are lined by normal mucous membrane, though about the bases of the gland-cells, towards the apex of the pocket, there is an increased number of small round cells. Immediately beneath the glands lies the muscularis mucosæ; this can be clearly traced around the pouch. External to the muscularis mucosæ, and separated from it by some loose connective tissue, is the true muscular coat. If this be traced towards the extremity of the pouch it is found to become thinned, its fibres being intermingled with fibrous tissue and interspersed with aggregations of lymphocytes. Outside this fibrous layer, which covers in the diverticulum, the wall of the gut is composed of loose connective tissue containing an inflammatory exudate becoming more cellular towards the peritoneal surface, beneath which are numerous extravasations of blood. The peritoneum itself is covered with a delicate layer of fibrinous lymph, in the meshes of which are entangled numerous lymphocytes."

False diverticula of the large intestine are of great pathological interest. Acquired diverticula are found in all

parts of the intestine, from the duodenum to the rectum. In the small intestines, though numerous, their clinical significance is of the slightest, for the thin fluid contents of the bowel do not readily become pent up in these little pouches. Gordinnier and Sampson (*Journ. Amer. Med. Assoc.*, vol. i, 1906, p. 1585) say that they have been unable to find a single case reported in the literature in which clinical symptoms have arisen from diverticula of the small intestine. In the large intestine the diverticula are not infrequently seen; the sigmoid flexure displays them more frequently than other parts of the colon. The false diverticula are always multiple; they are hernial protrusions of the mucosa and submucosa along a track in the intestinal wall weakened by the passage of a vessel. Once developed, they are prone to become places for lodgment of the solid, slowly-travelling intestinal contents. The various pathological conditions resulting from acquired diverticula are:

(1) Chronic inflammatory deposit in the walls of the bowel producing stenosis.

(2) Localised peritonitis which may lead to the formation of pus.

(3) General suppurative peritonitis, the result of perforation.

(4) Vesico-intestinal fistulæ, due to rupture of a localised abscess into the bladder.

(5) Inflammation and thickening and contraction of the mesentery, in which the pouches lie (mesenteritis).

(6) Carcinoma. Hochenegg has recorded the only case of cancer developing in a false diverticulum.

Case 6.—*Tumour of transverse colon and splenic flexure; ilio-sigmoidostomy.*—Mrs. W. S., æt. 62, seen with Dr. Holderness, December, 1903. The patient has suffered for seven months from flatulence, colic, constipation, and loss of weight. During the last three months she has twice been confined to bed for periods of three and four days respectively in attacks of subacute intestinal obstruction. In the last of these I saw her, and on examination I found the abdomen distended; coils of small intestine were seen contracting, and the cæcum, ascending, and transverse colon could be seen to distend and could be felt to harden. Loud rumbling noises were heard in the abdomen, and the patient told us that she had noticed these "bubbling" sounds for several weeks past. For the twenty-four hours before I saw her the patient had been very

nauseated and had vomited twice. The general condition was good. Shortly after my visit the obstruction yielded to a gravitation enema, the bowels being copiously relieved on several occasions, and the abdomen becoming soft and flaccid again. A diagnosis of malignant stricture of the splenic flexure was made, and the patient came to a nursing home for operation. On her arrival there the abdomen was hollow, the wall relaxed, and a tumour could be distinctly felt in front of the left kidney. When grasped between the hands the mass felt of the size of a cocoanut.

Operation (December 14, 1903).—An incision was made in the left linea semilunaris and the abdomen was opened. The transverse colon was found to be thickened and red; the descending colon and sigmoid flexure were pallid and empty. A large, excessively adherent mass was seen and felt occupying the left end of the transverse colon. The mass was hard, smooth, but irregular in shape; it was adherent in all directions to the abdominal wall, the stomach, the diaphragm, and to a few coils of intestine. Resection was impossible, and I had to be content with a short-circuiting operation. I secured the lowest part of the ileum and united it to the sigmoid flexure (ileo-sigmoidostomy). The distal limb of the ileum was narrowed by infolding sutures of linen thread.

The recovery of the patient was uneventful. Since the operation she has gained weight steadily; the tumour has entirely disappeared; nothing can be felt in the region it once occupied so fully. The bowels act well, and the patient's appearances justify her written statement that she "never felt better."

In this case I had no suspicion but that the tumour was carcinoma in a stage so advanced as to render resection mechanically impossible. But it is almost certain that I was mistaken, and that the tumour was a chronic inflammatory mass involving the colon. Its exact origin is quite uncertain.

In addition to these cases I have also seen, in consultation with Dr. Henry White, of Bradford, a patient, a man æt. 60 (?), upon whom colotomy was performed some years ago by one of the most distinguished of living surgeons, for intestinal obstruction due to a mass in the lower part of the sigmoid flexure or the upper part of the rectum—a mass which was supposed to be malignant in nature. The colotomy acted well, and to everyone's surprise the tumour gradually melted away, and eventually no trace of it could be discovered. The colotomy opening was then closed. Over a year later

a tumour had again formed, and intestinal difficulty was so great that the re-opening of the colotomy wound was under consideration. But persistence with enemata relieved the obstruction, and the bowels began to act regularly again. The tumour, which was still perceptible, gradually disappeared, and for the last few years the patient's condition has been perfectly satisfactory.

The six cases here recorded have only afforded four specimens, but each of these is of considerable interest. Of the three other cases I think it is safe to say that, in the first two, no one seeing the conditions disclosed by the operations would have hesitated for one moment to declare that the tumours were carcinomatous. In the last case the diagnosis of carcinoma of the sigmoid flexure was made, and treatment based thereupon was carried out by an acknowledged expert. Yet the tumour disappeared completely, re-appeared, and has again resolved.

The inflammatory tumours of the large intestine, excluding the tuberculous conditions, are, it would appear, far more frequent than we have supposed. The exact nature of the conditions present are not always the same. The inflammation may begin in and penetrate the mucosa; a false diverticulum may form and may be combined with a form of polypoid growth; or, finally, the inflammatory deposit may affect the peritoneal coat, chiefly or solely, leaving the mucosa supple and intact.

It is important to remember that the naked-eye appearance of the tumours in many of the cases is such that the mimicry of carcinoma is complete. Unless a careful microscopic examination is made differentiation is impossible. It is, accordingly, not unreasonable to suppose that in some, at least, of the cases of "cure" after colectomy for carcinoma an error has been made of the kind to which I have drawn attention.

X.—*A Case of Rupture of the Diaphragm.* By J. Paul Roughton, M.R.C.S. *Read December* 14, 1906.

Arthur T., æt 49, railway shunter, was admitted to hospital at 5.30 p.m. on October 27, having been squeezed between buffers, which caught him in the epigastrium.

On admission.—Careful examination revealed no lesion of any importance. The seventh and eighth cartilages of the ribs were separated from, and depressed below, the ribs. His temperature was 96° F. and his pulse 98. He was apparently recovering from shock. He complained of great pain in the back and precordia.

October 28, 2.30 *a.m.*—The house-surgeon examined him and found that the air entry over the left lung was deficient. He still complained of pain in the back and chest, which was increased on swallowing, and swallowing induced retching. He had been very restless. 9.30 a.m.: I found his condition much altered from that of the previous evening. He had had a bad night and still had the severe pain. His pulse had gone up to 130 and his respirations to 40. He was rather stupid and constantly moaned.

On examination.—The left hypochondrium was slightly retracted and the lower part of the abdomen moved only feebly with respiration. In all other respects the abdomen appeared to be normal.

Chest.—Movement on the left side was markedly impaired. On percussion the cardiac dulness was absent and the left side of the chest was hyper-resonant; the right side was normal as was the liver dulness.

On auscultation, above the third rib the breath-sounds had the character associated with a lung compressed by pleural effusion; below the third rib the breath-sounds were absent. On listening in the fourth interspace in the anterior axillary line and percussing the epigastrium with coins a well-marked bell-sound was heard; on swallowing water it could be plainly heard entering the stomach both in the epigastrium and over the left chest; an exploring syringe thrust into the chest withdrew air at a depth of two inches and blood at four inches, but not frothy blood. From these signs I inferred that the diaphragm was extensively ruptured and that the stomach had ascended into the chest, displacing and compressing the lung. I decided to operate, hoping to be

able to pull down the stomach, fix it firmly to the abdominal wall, and after the abdominal wound was closed, aspirate the air from the pleura which opening the abdomen would allow to enter into it.

On opening the abdomen by an incision through the left rectus the diagnosis was at once confirmed. I passed my hand high up into the chest, up to the compressed lung and past the heart. Unfortunately, the patient's condition was so grave as to force me to cease, and after pulling down the stomach I stitched up the wound. The traction on the stomach was followed by the passage of blood from the thorax into the abdomen, which at that time I attributed to the tear in the diaphragm.

After the operation the patient rallied somewhat and he had less pain; he was more collected, and he remained the same till 11 p.m., when he became worse and died at 1 a.m.

Post-mortem.—There was ecchymosis of the great curvature of the stomach, omentum, mesentery, and pericardium; the pericardium had an ounce or so of blood-stained fluid in it; the heart was normal. The left lung was completely collapsed, the chest being filled with about three-fourths of the stomach; the spleen had also entered the chest. The diaphragm was torn entirely away from its attachments between the ninth left rib and the ensiform cartilage, and, having contracted, formed simply a shelf between the thorax and abdomen. There was a tear in the spleen crossing its posterior border and extending about an inch and a half into its substance. This tear was doubtless the source of the blood which flowed from the thorax into the abdomen on traction of the stomach, the hæmorrhage, as the spleen was above the diaphragm, occurring in the thorax and not in the abdomen.

I thought it worth while to bring this case to the notice of the Society because of the unequivocal nature of the signs showing that the diaphragm was ruptured, or rather that the stomach was in the thorax, viz., the absence of cardiac dulness, the hyper-resonance of the chest, the compression of the lung above the third rib, the absence of breath-sounds, the conduction of the bell-sound from the epigastrium to the chest, and the ease with which the water, on swallowing, could be heard entering the stomach both in the epigastrium and over the chest.

I think it remarkable that so serious a lesion of the diaphragm presented no symptoms for so long a time after

the accident, and it was this fact which determined me to operate, as so large a rupture of the diaphragm did not at first embarrass him, and it was not until the attacks of retching had forced the stomach into the thorax that the patient's condition was alarming, and I therefore hoped that if I could only fix the stomach in the abdomen I might disregard the lesion of the diaphragm. On account of the absence of signs of blood in the peritoneum before the operation and its demonstrable absence on opening the peritoneum I overlooked the possibility of a rupture of the spleen, it not occurring to me that it might be bleeding into the pleura.

XI.— *A Case of Traumatic Hernia through the Diaphragm.* By JAMES BERRY, B.S. *Read December* 14, 1906.

FREDERICK P., a railwayman, æt. 19, while engaged in chalking a truck was struck in the back by another truck so that he was squeezed between the two buffers. He managed to fall clear of the line and to avoid being run over. He was picked up and brought to the Royal Free Hospital, where I saw him seven hours after the accident, on November 11, 1898.

He was then greatly collapsed and exceedingly pale. Over the upper part of the abdomen and the precordial region in front and in the corresponding regions behind, there were slight abrasions of skin and some subcutaneous bruising. The abdomen was not distended, measuring 28½ ins. in circumference at the umbilicus. No fracture of ribs could be detected. The heart was displaced considerably, the cardial dulness extending nearly two inches to the right of the sternum. The apex beat could not be felt. There was dulness over the whole of the left back as far forwards as the mid-axillary line and hyperresonance in front. There was no audible breath-sound over the left chest except at the upper and anterior part. Over the lower part of the right pleura there were also dulness and diminished breath-sounds. Respiration very shallow and forty to the minute. Pulse 130 and very feeble. Temperature on admission 96° F., rising to 98° F. in the course of the first twenty-four hours. The patient had passed urine naturally and there was no hæmaturia. The diagnosis made at this time was that of hæmopneumothorax due to laceration of the left lung and probably other viscera as well. It was suggested that, from the nature of the injury, the diaphragm might have been injured, but no definite diagnosis of this was made.

During the next three days the patient remained in much the same desperately bad condition. The signs of blood in the left pleura gradually increased, the dulness extending as far forwards as the anterior axillary line. The patient vomited occasionally and complained constantly of pain in the upper abdomen and in the precordial region.

On November 14 it was noted that the area of tympanites at the upper part of the left chest was less marked, but that

at the lower and anterior part there was a marked area of tympanites between the cardiac region and the area of dulness in the left lower axilla. This was continuous with the stomach resonance of the upper abdomen. The pulse at this time had fallen to 100, but was still very weak and often irregular both in volume and rhythm. Vomiting then began to be a prominent feature of the case, the patient frequently bringing up small quantities of brown coffee-ground material.

On November 15 the area of tympanites before referred to had extended higher up the front of the chest. It was also noticed that the left chest was somewhat retracted and that the area of cardiac dulness had shifted back nearly to its normal position. The circumference of the abdomen had diminished from 28½ to 24½ in. These physical signs seemed to indicate that the pneumothorax was disappearing and that its place was being taken by stomach and other abdominal viscera passing up into the thorax. The diagnosis of hernia of the stomach through a rent in the diaphragm seemed to be fairly clearly established. As the vomiting was becoming more urgent and strangulation seemed probable it was decided, after consultation with several of my colleagues, both medical and surgical, that, desperate as the man's condition was, he should be given what slender chance an operation might afford. The abdomen was accordingly opened in the middle line above the umbilicus. A rent in the left posterior part of the diaphragm large enough to admit the hand was found. Through this most of the stomach and transverse colon, the whole of the great omentum, and the upper part of the left kidney had passed. The spleen had been cut into two nearly equal halves, the upper portion lying free in the left pleural cavity, which contained also a large amount of fluid blood. The herniated viscera, which were moderately constricted by the edges of the opening in the diaphragm, were drawn back with some little difficulty. The rent in the diaphragm was then closed, partly with suture and partly by utilising the left lobe of the liver to block it up. The operation lasted half an hour and the patient died very shortly after its completion—at 1.30 a.m. on November 16.

The *post-mortem* showed little more than has already been mentioned. The left lung was completely collapsed and the left pleura contained much blood.

The difficulty of exact diagnosis in this case was considerable, especially as the patient's general condition through-

out was so bad. The original principal injury was no doubt the damage to the left lung causing hæmopneumothorax. It seems to me that as the pneumothorax became absorbed the abdominal viscera were gradually and slowly forced up into the thorax, probably about the third day after the injury. The physical signs of pneumothorax were then gradually replaced by those of hernia of the stomach. The large size of the opening in the diaphragm prevented anything like tight strangulation of the herniated viscera. Vomiting, therefore, was not severe until the herniated portion of the stomach became greatly distended and congested. I take it that the coffee-ground material that was vomited came chiefly from the distended and congested portion of the stomach within the thorax.

It may be thought by some that operation ought to have been undertaken sooner. But it must be remembered that owing to the presence of other severe injuries, such as the rupture of the spleen and the laceration of the lung and the extensive hæmorrhage that accompanied these injuries, the man's condition was at no time such as to justify the performance of a large operation, except under the most pressing conditions. It was the nature and persistence of the vomiting that eventually compelled me to intervene. I venture to think that the vomiting of considerable quantities of coffee-ground material is, in itself, an important indication of strangulation of the stomach. In the only other case of strangulation of a diaphragmatic hernia of the stomach that has come under my observation this same kind of vomiting was a most marked feature of the case. This case was under the care of a surgeon at St. Bartholomew's Hospital many years ago. The hernia was first discovered in the *post-mortem* room.

XII.—*Acute Peritonitis with Fat-necrosis: no Disease of the Pancreas found.* By JOHN FAWCETT, M.D. *Read November* 23, 1906.

ANNIE S., æt. 59, was admitted into Guy's Hospital on October 15, 1905, under the care of Dr. Hale White, for severe abdominal pain and vomiting which had commenced suddenly on the previous day. The pain was at first located in the upper part of the abdomen; the patient was sick in the evening of October 14, and the vomiting continued at intervals during the following day.

The only feature of interest recorded with regard to her past history was that six months before admission she had had two or three attacks of pain in the upper part of the abdomen, which she described as being of a similar, though less severe character, than the present one.

Condition on admission.—Temperature 95° F., pulse 88, respirations 28. The patient was in great pain, constantly calling out for something to relieve it. The abdomen was neither distended nor retracted and moved well with respiration; it was not rigid but was extremely tender on palpation in the upper part. There was a resonant note everywhere on percussion.

The urine was free from abnormal constituents.

The temperature remained subnormal throughout and the pulse-rate did not rise above 92 per minute.

During the subsequent twenty-four hours the pain became somewhat less in severity and the sickness ceased, but after this interval of time the patient began to vomit again, and about three hours later the vomit was said to be "stercoraceous." Laparotomy was then performed (October 16). A small quantity of a brownish fluid was present in the peritoneal cavity; several areas of fat-necrosis were seen in the great omentum, and below the lesser curvature of the stomach a small quantity of extravasated blood; no other pathological changes were detected.

The patient gradually sank after the operation and died on October 17.

Autopsy.—I performed the *post-mortem* examination on October 18 and found a recent generalised peritonitis present with a quantity of fluid having a bile-like colour in the peritoneal cavity.

There were a number of adhesions between the stomach, the duodenum and the great omentum in front, and the pancreas posteriorly. These adhesions were probably of some age for they required considerable force to separate them.

A wide-spread fat-necrosis was present on the great and the lesser omentum, and over the front of, and around, the pancreas; the necrotic areas were pale yellow in colour. There was a recent hæmorrhage, the size of a walnut, in the gastro-hepatic omentum.

The pancreas was firm and hard to cut; no abnormal changes were visible in it.

The gall-bladder appeared to be quite healthy and also the bile-ducts; in the interior of the gall-bladder were three calculi, the largest the size of a hazel nut, and the smallest, which was loosely fixed in the orifice of the cystic duct, the size of a pea. The stones were composed of cholesterin and pigment. The common bile-duct appeared to be somewhat dilated, measuring just over ¼ in. in circumference; its lining membrane was healthy and there were no signs of any previous damage to the duct. All the other organs of the body were healthy.

Bacteriological examination.—Cultivations made by Dr. Eyre from the spleen and from the blood in the heart exhibited a growth of *Bacillus coli communis* only.

Microscopical examination.—The pancreas appeared to be quite healthy, the glandular epithelium staining well and exhibiting a normal appearance, and there was no small-celled infiltration of the viscus.

Remarks.—Although in the majority of cases fat-necrosis is associated with disease of the pancreas, yet there are some cases in which no lesion of that organ is discoverable; a number of these are now on record. Opie refers to those published by Flexner and Fraenckel. Haffner in the *Münchener med. Wochenschrift*, li, 1904, relates a very similar case to the one here recorded of disseminated fat-necrosis, without any lesion of the pancreas, in which the symptoms suggested perforation of a hollow viscus such as the stomach. In the Guy's Hospital *post-mortem* records during the past six years there are notes of three cases in which fat-necrosis was found without any obvious disease of the pancreas, although special attention was paid to this organ; in none of these three were microscopical sections of the pancreas examined, but notwithstanding this the cases are of interest and are here briefly recorded.

Case 1.—Eliza R., æt. 33, was admitted under Dr. Washbourn on July 14, 1901, for acute abdominal pain, which had commenced on the previous day. The abdomen was opened by Mr. Lane twelve hours after the onset of the attack but no cause was found to account for the symptoms.

Autopsy (July 15).—A small quantity of brownish fluid was found in the peritoneal cavity. There were numerous areas of fat-necrosis in the very fat omentum, in the appendices epiploicæ, and on the intestinal coils. In the region of the pancreas the tissues were stained by a deposit of altered blood. The pancreas was of firm consistence and exhibited no change whatever to the naked eye; there was no hæmorrhage into its substance. The gall-bladder was large, its walls thick, and in its interior were a number of small pigment-calculi with cholesterin on their surface.

Case 2.—Sarah E., æt. 45, was admitted under Dr. Hale White in 1901 with enlarged cervical glands which proved to be sarcomatous. She died of hæmatemesis, having previously suffered from epistaxis, bleeding from the gums and other places.

Autopsy (October 2).—A healed duodenal ulcer was situated at a distance of 3 cm. below the pylorus and was firmly attached to the pancreas. There were many areas of fat-necrosis scattered over the visceral peritoneum, especially numerous in the region of the pancreas, behind the stomach, and in the great omentum. The pancreas appeared to be perfectly normal.

Case 3.—J. R., æt. 43, a labourer, was admitted under Dr. Shaw on August 16, 1902, for an attack of acute abdominal pain which had set in suddenly on the previous day. Laparotomy was performed and a perforated ulcer was found on the posterior surface of the stomach; this was closed by suture. The patient died on August 17. No areas of fat-necrosis were seen at the time of the operation.

Autopsy.—There was a recent acute peritonitis, and in the fat of the great omentum a few opaque yellow areas of fat-necrosis. The ulcer was of the acute type. The gall-bladder was empty. The pancreas appeared to be perfectly healthy.

Opie, in his work on *Diseases of the Pancreas*, agrees with the generally-accepted view that fat-necrosis is caused

by the pancreatic ferment invading the tissues and exerting its fat-splitting action upon the living fat-tissue. He alludes to the fact that Flexner has found the ferment in the necrotic areas, and says that "fat-necrosis is always a consequence of pancreatic disease."

To accept this statement as it stands renders an explanation of such a case as the one here recorded somewhat difficult. The setting free of "steapsin" is almost certainly the cause when the pancreas is damaged, but in the absence of this one must at any rate keep an open mind as to the possibilities of a fat ferment being generated elsewhere than in the pancreas, and that such a ferment may arise from the action of bacteria. On the other hand it is probable that we do not yet know all the conditions under which a fat-splitting ferment is set free from the pancreas; when we do possess a more complete knowledge of the subject we may find that the formation of the necrotic foci is, after all, dependent on the pancreatic ferment. It is, I think, true that even in the cases where the pancreas appears to be healthy the greater number of the necrotic patches are in the neighbourhood of the pancreas, and it may be that obstruction to the pancreatic duct or interference with the blood-supply to the gland is sufficient, in some cases, to set the ferment free without producing any lesion of the gland itself, which is recognisable either by the naked eye or microscopically.

Under present conditions of knowledge it can hardly be said that the pancreatic ferment theory is a satisfactory explanation of cases in which fat-necrosis is found in the adipose tissue of the pericardium or in the subcutaneous fat. Also it is equally unsatisfactory to suggest that, inasmuch as in some of the cases gall-stones have been found, the fat-necrosis in these cases is the result of compression of the pancreatic duct by the calculi. This may explain some cases but certainly not all. In the case I have related, although there were gall-stones in the gall-bladder the duct was not compressed. In Case 2, from the Guy's records, it is possible that the pancreatic duct may have been obstructed by the adhesions between the healed duodenal ulcer and the pancreas, and in Cases 1 and 3 the gland *may* have been damaged at the operation. This explanation would not suffice in the case I have related for the necrotic foci were seen at the operation. If an injury of this kind does lead to fat-necrosis it must be exceedingly rare, for otherwise, with the large number of laparotomies now performed for the treatment of various

conditions in the region of the stomach fat-necrosis would probably be found more frequently.

From the clinical point of view the interest of the case lies in the fact that there is still another condition which simulates a peritonitis due to perforation, of which one of the most prominent pathological features is the presence of a widespread fat-necrosis. That this condition is not so exceedingly rare is shown by the cases which have occurred at Guy's Hospital here recorded, and also in the careful report of a similar case by Haffner previously referred to. Whatever the cause may ultimately prove to be, the acute abdominal symptoms manifested are probably due to the early involvement of the peritoneum, and not directly to the necrotic fat-deposits.

XIII.—*Case of Acute Pneumonia, with Abscess of the Lung and Acute Endocarditis (Endocarditis pneumonica).* By Francis H. Hawkins, M.D. *Read January* 11, 1907.

WILLIAM J. G., æt. 51, labourer, was admitted under my care on April 26, 1906, with the following history: About the first week in March the patient had influenza, from which he recovered. On April 19 he was suddenly seized with severe pain in the abdomen and the right side of the chest; this was followed by cough and a good deal of blood-stained expectoration. On April 24 he had diarrhœa, and on the following day he vomited.

State on admission.—Patient is a well-developed, somewhat spare man. His face is flushed, eyes staring, and he is lying on his back.

Examination.—The respiratory movements are mainly abdominal (34 per minute). The left half of the thorax moves slightly better than the right, but neither side moves freely.

Right side (anteriorly).—The percussion note is dull from the clavicle to the fourth rib; below it is resonant till merging into the liver dulness. The breath-sounds over this dull area are indeterminate in character. No adventitious sounds are heard.

Posteriorly.—The percussion note is dull over the supra- and interscapular regions. The breath-sounds over these regions are bronchial in character.

The percussion note over the base of the lung is resonant, and numerous râles are present at the base and over the axillary region. Over the left side there was nothing abnormal to note.

The sputum is viscid, of a rusty colour, and, in parts, slightly tinged with blood.

Heart.—Apex normal; no murmur is heard.

Pulse.—104, regular.

The tongue is dry and brown. Bowels moved once; stool loose. Temperature 103° F. Urine, sp. gr. 102; albumen, a trace.

April 27.—During the night the patient was very delirious, and he was constantly trying to get out of bed. When seen

during the day there was well-marked tubular breathing over the right infra-clavicular region. The temperature (four-hourly) at 3 a.m. was 102·6°, at 7 a.m. and at 11 a.m 101°, at 3 p.m. and 7 p.m. 100°, and at 11 p.m. 101·2° F. The bowels were open four times; stools liquid.

April 28.—Patient was still delirious. Temperature at 3 a.m. was 100·2°, at 7 a.m. 99·8°, at 11 a.m. 98·4°, at 3 p.m. 100·8, at 7 p.m. 99·2°, and at 11 p.m. 99° F. Bowels moved once.

April 29.—The temperature at 3 a.m. was 96° F. The patient was now quite sensible. The breath-sounds over the infra-clavicular region were tubular in character, and fine crepitations were heard.

The temperature on May 1 was 98° F.

Further report of the case.—The dulness over the right infra-clavicular region did not disappear, but extended somewhat lower down. The breath-sounds over this region varied in character, some days being tubular, and at other times of an indeterminate character. Crepitations, and later coarse râles, were heard. From May 1 the temperature was higher each day, and on the 6th it was 105·4° F. at 3 a.m.; at 11 a.m. it fell to 102° F., and the patient then perspired profusely. On May 7, at 3 a.m., the patient had a rigor and vomited; the temperature was 104·8° F.; after sponging it fell to 101·6° F., rising at 11 a.m. to 104·6° F. At 3 p.m. it was 104·4° F., but at 7 p.m. it had fallen to 96° F., and the patient again perspired profusely. On the 9th the patient lost control of the sphincters, and the temperature, notwithstanding the use of the ice-cradle and sponging, kept up to 104·8° F. On the 10th, at 5.10 a.m., the patient had a convulsive seizure, followed by semi-coma, which gradually became complete, and he died at 3 p.m. During this time no cardiac murmur was heard. The tongue was dry and covered with a brown fur. The bowels moved daily from two to four times. The albumen in the urine was never more than a trace.

Necropsy.—Thorax: The upper and middle lobe of the right lung were adherent to the chest-wall by recent adhesions.

Right lung: The right upper lobe was in a state of grey hepatisation, and contained three abscesses; two were deep seated, and about the size of a large walnut, the third was superficial, near the anterior border on the level of the third and fourth cartilages, and was of the size of a hen's egg; all three contained pultaceous greyish pus, and had a distinct

lining membrane. The lower lobe of the right lung and the whole of the left lung were œdematous.

Heart: At the base of the posterior aortic cusp was a recent fibrinous deposit, which, on being removed, showed an ulcerated surface about the third of an inch in diameter.

The liver was enlarged and soft. The spleen was much enlarged and its substance almost diffluent. The kidneys were enlarged. There were no infarcts in any organ.

Remarks.

I may, perhaps, first mention that I have myself met with one case of abscess of the lung following acute pneumonia other than that just recorded—a man between thirty and forty, in whom the abscess was situated in the right lower lobe and undoubtedly communicated with a bronchus, and was thus evacuated. The case was very protracted, but eventually ended in recovery. The notes of this case are unfortunately not now to be found.

Abscess of the lung as a complication of acute pneumonia is undoubtedly rare. Wilson Fox thought it was probably more rare than gangrene. Von Leyden, writing on pneumonia, says: "Termination in abscess is quite rare, although frequent enough to require consideration." West, in his book on 'Diseases of the Organs of Respiration,' writes: "Abscess after pneumonia is extremely rare." Of 1165 cases in the medical statistics of St. Bartholomew's Hospital abscess occurred in two cases only, one a boy, æt. 11, and the other a man, æt. 30. Sturges and Coupland also refer to abscess of lung following pneumonia as being rare.

During a period of ten years 1006 cases of acute pneumonia (687 males, 319 females) were admitted into the Middlesex Hospital. Necropsies were made on 149 cases and in no instance was an abscess found. In an analysis of 708 cases of acute pneumonia in St. Thomas's Hospital during a period of eleven years (made by Dr. Hector Mackenzie and the late Dr. Hadden) necropsies were made on 128, and in no case was there an abscess of the lung. Aufrecht, in 253 fatal cases (202 men, 51 women) found 3 cases of abscess. Lebert states that he has met with 4 cases; the cavities were small, varying between the size of a hazel nut to that of a walnut, and Osler found 4 cases of abscess in 100 autopsies clothed with an organised pseudo-membrane.

Louis Grisolle and Andral have each only observed 1 case; the latter states, "as for ourselves it has never fallen to our lot more than once to see a real abscess after a pneumonia at the La Charité. It was in the case of a man who died on the nineteenth day of inflammation of the lung. The middle and lower lobe of the right lung were in a state of grey hepatisation; towards the middle part of the lower lobe there was observed nothing but a sort of pap of a dirty grey colour, in the centre of which was found real pus which exhaled no odour, the pulmonary tissue around which was at first very soft and, as it were, broken down; gradually recovered a greater consistence. No particular sign had disclosed to us during life the existence of this abscess." Andral refers to a specimen presented to the Royal Academy of Medicine by Dr. Honori where the abscess cavity had smooth parietes, the cavity being the size of a large apricot. Grisolle, in 29 cases collected from different authors, found that in most cases the abscess had an irregular sinuous outline and that the walls were bounded by shreds of tissue; only in rare cases was an abscess membrane present. Aufrecht considers that when such a lining membrane is present the abscess must have occurred in a very late stage of the process.

It would appear that most abscesses are found in parts which are the seat of grey hepatisation, and they are proportionally more common in the upper lobes; thus in 26 cases collected by Grisolle 12 were in the upper lobe, 9 in the lower, 1 in the middle, and in 1 case they were multiple.

Rare as is abscess of the lung complicating pneumonia such associated with acute pneumonia and acute endocarditis is still more rare. I have myself not been able to find any such case recorded or even referred to in literature. Indeed, acute endocarditis is, in my experience, not commonly met with in pneumonia. Aufrecht states that he has himself seen but a single case of acute endocarditis affecting the aortic valves in the course of pneumonia. His view is that such varies as to time and location. Grisolle also speaks of it as a rare complication, and the committee of collective investigation in their report of 1000 cases found endocarditis only once mentioned. Professor Osler's experience, however, differs, for in 103 necropsies on cases of lobar pneumonia, acute endocarditis was noted in 16—over 15 per cent. Of these 11 were of the malignant type. In 6 of these cases the left lung was involved, in 5 the right, in 4 the

upper lobe was affected, and in 7 the lower. Professor Osler quotes Bouillard as having pointed out that pneumonia is not infrequently complicated with endocarditis, and Bouillard thought that in a third or fourth of the cases in which there was left-sided pneumonia there was inflammation of the serous membranes of the heart. Professor Osler's experience differs from this; he found that of 36 cases the right side was affected in 26, and the left in 10 only.

Now, in reading through the notes of 149 necropsies on cases of acute pneumonia in the published report of the Middlesex Hospital I find 4 cases in which endocarditis was present:

(1) A male, æt. 39, with pneumonia of the left lung; there was recent endocarditis on cusps of the mitral valves.

(2) A male, æt. 25, with double pneumonia; there was "one small recent vegetation on mitral valve."

(3) A male, æt. 5½, with pneumonia of the upper and middle lobe of the right lung; "Two patches of vegetation on cusps of tricuspid valve with some ulceration.

(4) A male, æt. 54, with pneumonia of the left lung. There was vegetative endocarditis of aortic valves. In the 128 necropsies recorded in the analysis of cases made in St. Thomas's Hospital, to which I have before alluded, one case of ulcerative endocarditis of the tricuspid valve was met with.

These cases are of interest, showing that both the vegetative and the ulcerative forms of endocarditis may be met with. Weichselbaum (quoted by Aufrecht) states that endocarditis pneumonica is found in two forms—the ulcerative and the verrucose. The former, he says, is far more frequent: in his cases the ulcerative variety was noticed six times and only once did the verrucose form occur. Heschl, Netter, and Weichselbaum are all agreed that the aortic valves are more frequently affected than the mitral; the latter found the aortic valves affected five times and the mitral only twice; neither mention the tricuspid valves being affected.

In 6 cases (4 from the Middlesex Hospital published reports, and 1 from the analysis of cases to St. Thomas's Hospital, and the case I now record) I find the aortic valve affected in 2 cases, the mitral valve in 2, and the tricuspid in 2. So far as I am aware these cases have not been referred to before.

In the case I have brought before you no bacteriological examination was made, but, nevertheless, I venture to describe

it as a case of endocarditis pneumonica. Weichselbaum has demonstrated the *Diplococcus pneumoniæ* upon the growth from the valves, and Netter in 9 cases of endocarditis pneumonique found in 7 cases the same bacteria as in the pneumonic lung.

I may just refer to the fact that in the case now recorded no cardiac murmur was heard; it was constantly listened for, as the case seemed to me to be one of ulcerative endocarditis complicating the pneumonia; and it is of interest to note that in the cases recorded by Professor Osler in his Gulstonian lectures no murmurs were heard.

XIV.—*A Fatal Case of Myxœdema, with changes in the Parathyroid Glands.* By DAVID FORSYTH, M.D. *Read January* 11, 1907. *With microphotographs. Pls. IX and X.*

MR. PRESIDENT AND GENTLEMEN,—The modern treatment of myxœdema has so affected the mortality of the disease that opportunities of examining subjects *post mortem* are by no means common. While this result is wholly satisfactory to us as clinicians, from a pathological point of view it is perhaps regrettable, because the work of recent years suggests that the pathogeny of myxœdema is more complex than was at one time believed. The attempted production of the disease in animals has led to results more contradictory than would have been anticipated. The thyroid has been removed, and the chronic symptoms of myxœdema have followed; it has been removed, and acute nervous symptoms leading to speedy death have resulted; it has been removed with no untoward accident being produced. These results raise a question as to whether myxœdema is attributable to the loss of the thyroid function alone, and the doubt is fostered by the fact that in some cases the administration of the gland extract fails in its results. The whole question has been still further complicated by the supposition on the part of many that the parathyroid glands possess a function of prime importance which is intimately bound up with that of the thyroid. To what extent (if any) these small bodies are concerned in the production of myxœdema is not settled; in fact, as far as I am aware, no account of their condition in atrophy of the thyroid has been published.

The particular case I am bringing to your notice this evening derives its interest, not so much from its clinical features as from the *post-mortem* findings, yet I feel no excuse is needed for submitting it to this purely clinical gathering. The thyroid gland can, without exaggeration, be looked on as the peculiar property of our Society, seeing that its relation to myxœdema was here established. Under these circumstances an account of the parathyroid glands will not be without interest to-night.

The patient with whom we are concerned was under observation only a short while before death, and the notes of her condition are brief. She was admitted into Guy's Hospital

under Dr. Hale White, and to him I am indebted for access to the clinical report, of which the following are the salient points:

N. P., æt. 58, married, was admitted on March 19, 1906, for "general debility." Her father died of heart-disease at the age of forty-nine; her mother lived till eighty-four, when she died of old age, and in neither was there any history of thyroidal trouble. The patient bore three children, all of whom are alive and well, the eldest being twenty-six and the youngest twenty. There is no history of any family hereditary disease.

The previous illnesses include none more serious than scarlet fever and German measles in childhood. The outstanding feature of her adult life appears to have been intemperance.

The mental state of the patient disabled her from rendering any trustworthy account of her illness, and the details were obtained from the husband. By him the disease was dated back four years, beginning insidiously. Even before that date, however, comment had been made on the inactivity of her habits, and on her marked disinclination to expose herself to the open air.

For three years the progress of the disease was slow, but twelve months ago she was unable to get upstairs without assistance, and since then she had grown more and more inactive. Two or three months before admission the disease began to make rapid strides. Her mind became affected and she grew child-like. What little power of locomotion she still retained deserted her, and she was unable to walk. Persistent pain settled in the back of the neck. She lost control of both bladder and rectum. Her teeth loosened and fell out without preliminary decay. Her hair, not only on the scalp, but from the eyebrows too, was shed, and she rapidly sank into a helpless and apathetic state.

On admission the temperature was 96° F., pulse 86, and respirations 20. The patient was noted as a little woman, with a complexion suggesting the yellow tint of pernicious anæmia. She was singularly dull and listless, and the difficulty of communication with her was not lessened by a certain suspiciousness in her attitude towards others. To questions her replies were delayed, and were then given in slow, deliberate manner, with thick articulation. As already mentioned, she was unable to supply details of her past history.

She lay still in bed on her back, and complained that

movement of her legs was painful, and of her head very painful. Her arms she used in a slow and lumbering fashion. No teeth remained in her jaws, and much hair had fallen out from the scalp, but the baldness was more marked in the eyebrows. The tongue was clean, but abnormally large. The skin over the whole body was dry, very tough, and yellow, and the slightest touch appeared to give pain, especially on the soles of both feet. The left ankle was red and swollen, and the subcutaneous tissue about it pitted on pressure. The hands were cold and spade-shaped, and the thyroid appeared atrophied. The pupils reacted to light and accommodation. The calves of the legs were not tender, and there was no paralysis, though the grip of the hands was weak. Knee-jerks normal; flexor plantar reflex; loss of control of the bladder. The respiratory and circulatory systems presented nothing abnormal. The abdomen was large—35 in. at the umbilicus—but no ascites had formed. The spleen was not palpable, but the liver was felt 2 in. below the costal margin. An examination of the blood showed red corpuscles 1,700,000 (34 per cent.), white corpuscles 5000, hæmoglobin 30 per cent.

The progress of the case was from the first towards a fatal end. Her mind wandered considerably, and her memory was unretentive of the most recent events. She grew more and more noisy, especially at night, and died on April 2—a fortnight after admission. The treatment employed consisted of five grains of dried thyroid extract three times a day and a mixture containing potassium iodide. The former was omitted for a couple of days as it was suspected to be in part responsible for the delirium.

Post-mortem examination made twelve hours after death.—There were old-standing adhesions between the lobes of the lung and between the lungs and diaphragm. The apex of the left lung contained a calcareous deposit, and its base showed hypostatic pneumonia. The heart, which weighed 384 grms., presented some fatty infiltration of the myocardium and hypertrophy of the left ventricle. The anterior cusp of the mitral and all the cusps of the aortic valve were slightly atheromatous, as also was the aorta itself, thoracic and abdominal. The liver slightly fatty, and the kidneys with some interstitial nephritis, complete the list of organs other than the thyroid and parathyroid which were affected.

Thyroid gland.—A detailed examination was made of the structures in the neck. The whole of the soft tissues anterior

to the prevertebral muscles and deep to the sterno-mastoid were removed *en bloc*, including everything from the tongue to the superior mediastinum. These were carefully dissected, and every gland discovered was retained for microscopical section. The thyroid itself in the usual position was remarkably small, pale, and indefinite in outline, merging into the surrounding connective-tissue. The isthmus was represented only by a fibrous band spanning the trachea. After dissecting the gland from its attachments, its lobes were seen to have none of the characters (colour, consistency, etc.) of the healthy thyroid and, except at the lower poles, none of the usual contour. The whole organ was a thin sheet of fibrous tissue wrapped round the front and sides of the trachea.

In close connection with the right lobe were two parathyroid glands—one lying on the lower part of the lateral surface, the other a short distance inferior to the gland. On the left side were four parathyroids, two large and two minute. Of the former, one lay on the lateral surface of the gland, the other just above the point of entry of the inferior thyroid artery. Close to this last were the two minute parathyroids.

All six glands were imbedded in much fat and rested on the thyroid without being depressed into its surface. The four largest were elongated ovoids from ·5 to 1 cm. in length, the two smallest mere pin-heads. All were fairly soft in consistency and of a dirty, yellowish-brown colour.

On the left side below the thyroid gland were two pale brown bodies, of which the note made at the time of the autopsy was "tough, almost fibrous." The nature of these will be discussed below.

In addition to these a large number of glands were examined microscopically, but all proved to be lymphatic.

Microscopical appearances of the thyroid and parathyroid glands.—The thyroid presented the well-known features characteristic of myxœdema. There was an almost complete disappearance of gland tissue which had been supplanted by fibrous tissue. Here and there were collections of small round cells and in other places clumps of degenerated thyroid tissue. In these there still remained a suggestion of the normal follicular structure, and many of the altered vesicles enclosed small drops of colloid. The arteries of the gland showed the marked thickening of their coats, which has been frequently observed in other cases.

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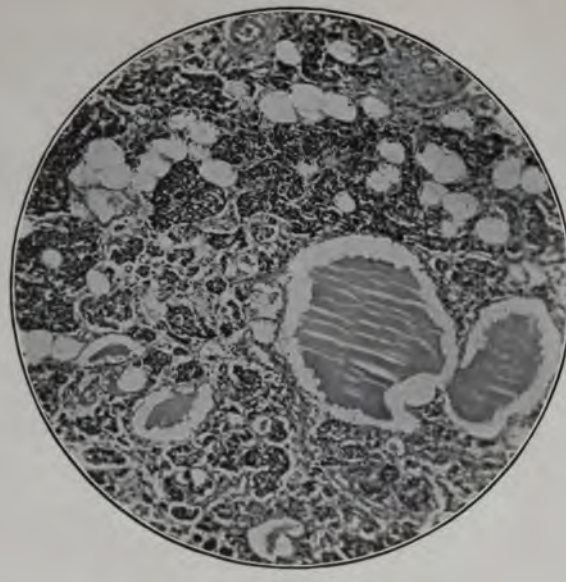

FIG. 1.

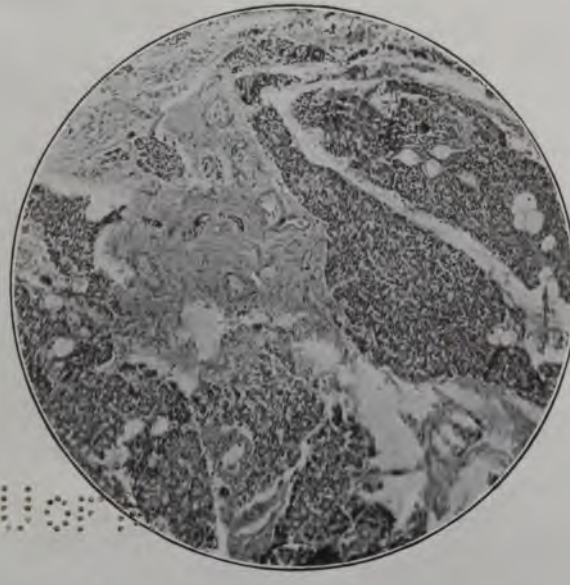

FIG. 2.

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PLATE IX.

Illustrating Dr. Forsyth's paper on "A Fatal Case of Myxœdema."

FIG. 1.—Parathyroid from fatal case of myxœdema showing many lymphatic vessels containing colloid secretion. The largest vessel has a tributary about to open into it. Above are two arterioles with thickened walls. Throughout the field the cells are isolated into small clusters by the increased connective tissue strands. Zeiss A.

FIG. 2.—Another parathyroid from the same case showing increase of connective-tissue trabeculæ whereby a lobulated appearance is conferred on the gland. Zeiss A.

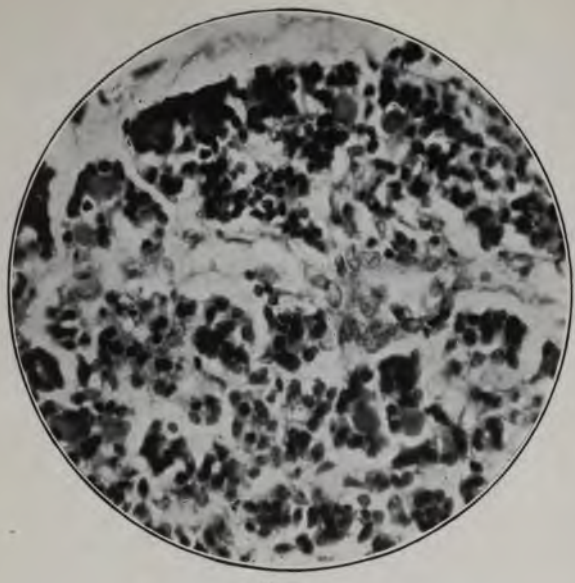

FIG. 3.

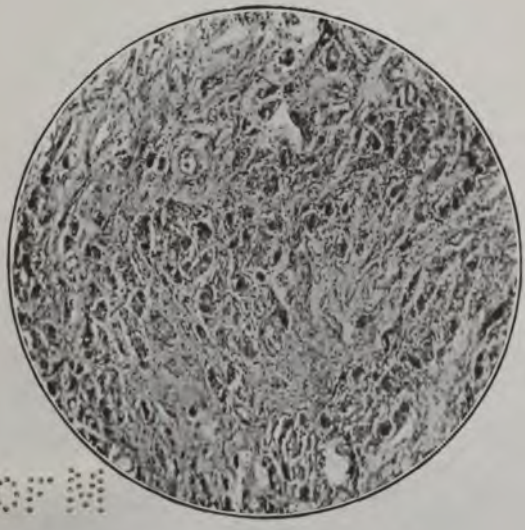

FIG 4.

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PLATE X.

Illustrating Dr. Forsyth's paper on "A Fatal Case of Myxœdema."

FIG. 3.—Another parathyroid showing numerous drops of colloid lying in primitive vesicles. Zeiss D.

FIG. 4.—One of the fibrous bodies mentioned in the text, showing a fairly advanced stage of fibrosis. Some glandular structure with drops of colloid persists. Zeiss A.

The parathyroid glands were compared with those taken from a large number of other subjects, and certain points of difference were apparent. The normal gland, it will be remembered, is made up of solid masses of closely-packed polyhedral cells, the groups being supported by delicate trabeculæ of connective tissue. These cells are arranged, for the most part, without order, but occasionally they show a tendency to group around a common centre, and less frequently a completely formed acinus results, the lumen of which is filled with colloid secretion.

The special features wherein the glands from this case of myxœdema appeared to differ from the ordinary glands were (Pls. IX and X), firstly, in the arrangement of their cells; secondly, in the quantity of their secretion; and thirdly, to a less extent, in the stroma and in the walls of the blood-vessels.

The arrangement of the cells was unusual, owing to the marked tendency they showed to form small vesicles. In contrast to some parathyroids where a more or less prolonged search is required before an example of this acinar arrangement is discovered, in these specimens there were few fields without acini, and many fields contained a dozen or more. In each vesicle the epithelium was a layer of cubical cells. The resulting general appearance of the glands was much more suggestive of ordinary thyroid tissue than is usually the case. As regards the features of the individual cells, there was no point to be noticed in either nucleus or protoplasm which could not be frequently observed in other parathyroid glands.

The colloid secretion was copious. With few exceptions all the vesicles contained this material, and drops of it lay between the clumps of unarranged cells. The lymphatics of the gland were distended with the same secretion, and on serial section could be traced towards the surface, uniting to form large channels, which conveyed the colloid away from the gland. After examining some hundreds of parathyroids in none do I remember such vigour of secretion as in these.

The slight change mentioned as present in the stroma consisted in some increase of the connective tissue. Certain of the trabeculæ were thickened, and in parts of the glands there was a delicate intercellular mesh of connective tissue. In some of the glands there was, further, a slight thickening of the walls of the arterioles.

The two fibrous bodies, to which reference has already been made, proved, on section, to possess a glandular structure

with a fairly advanced condition of fibrosis—less advanced than in the thyroid, more advanced than in the parathyroids. From the remains of the glandular elements it is not easy to decide whether the bodies were originally accessory thyroids or parathyroids, for the details of structure might represent either the one or the other.

In conclusion, it remains to offer an explanation of the changes in the parathyroids. It is not possible, however, in the limits of this paper to discuss the various interpretations that might be put forward, and I shall content myself with offering what seems to me the correct solution.

I do not think that we should regard the changes in the arrangement of the cells and in the amount of secretion as pathological; rather are they to be interpreted as signs of excessive activity of the glands. The particular reason we advance for this unusual secretion will depend on the view we take of the physiological importance of the parathyroids —whether we regard them as glands *sui generis* or as glands possessing a true thyroidal function. Accepting the latter hypothesis as the conclusion most in accord with available evidence, it seems reasonable to suppose that these changes represent an attempt on the part of the parathyroids to supply the deficiency of colloid left by the atrophy of the thyroid itself.

The increase of connective tissue, though not excessive, was considerably more marked than I have seen in any other human gland, or, with one exception, in the parathyroid from any mammal or bird. Although five out of the six parathyroids were directly continuous with the fibrous capsule of the thyroid, this factor alone will not account for the alteration, because the sixth parathyroid, which lay inferior to the main gland, was quite as affected as its fellows. I look on the increase of fibrous material as pathological, in which case it must be regarded as an early stage of the fibrosis, which had reached so advanced a condition in the thyroid.

In brief, these changes show that the parathyroids have been stimulated to supply, in part, the deficiency of colloid, but that at the time of death they were being overtaken by the same fibroid degeneration as had invaded the thyroid.

XV.—*On Tetany associated with Dilatation of the Large Intestine in Children.* By FRED. LANGMEAD, M.D. *Read January* 11, 1907. *With Pl. XI.*

IT was my privilege last year to read before this Society the notes of the case of a child, in whom repeated attacks of tetany were associated with dilatation of the sigmoid flexure. I ventured at that time the opinion that the association was not merely a coincidence, but that the dilated loop of large intestine was analogous to the dilated stomach in gastro-tetany—in other words that the tetany was the result of the absorption of toxins generated in the stagnant contents of the bowel. Since then three somewhat similar cases have come to my notice which seem to remove all doubt as to the truth of that conjecture.

CASE 1 is that already reported in the *Transactions* of this Society, vol. xxxix, and so I will only briefly refer to it. It was that of a boy, æt. 6, who between November, 1903, and March, 1905, was admitted four times into the Hospital for Sick Children, Great Ormond Street, under the care of Dr. Garrod, suffering on each occasion from an attack of tetany. It was noticed that each attack was accompanied by the evacuation of yeasty, frothy, porridge-like stools, and by distension of the abdomen. By palpation and percussion it was evident that a large, hollow viscus was situated on the left side of the abdomen, reaching from the left iliac region up to the epigastric and left hypochondriac regions. All treatment was ineffectual until lavage of the large intestine by means of a rectal tube was resorted to, when on each occasion improvement was initiated and led to complete disappearance of the tetany. Soon after being discharged for the fourth time he was re-admitted for tetany and diphtheria, which proved fatal. At the *post-mortem* examination the hollow viscus recognised during life was identified as the sigmoid flexure, which was very much dilated, occupying the greater part of the left half of the abdominal cavity and accommodating about 13 oz. of fluid.

CASE 2 is that of a boy, æt. 3 years and 2 months, who was admitted into the Hospital for Sick Children also under the care of Dr. Garrod, by whose kindness I am allowed to use these notes, on May 10, 1906. His mother said that he had

been passing whitish motions three or four times a day for about six months, and that on four different occasions the hands had been, as she put it, "drawn up" for a week or two at a time. The first attack had occurred when he was nine months old, the last had been present since four days before admission. He was the youngest of nine children, and had always been backward; he had only just begun to talk, and had never been able to stand or walk. His state on admission was very like that of the patient already reported on, the similarity in appearance being especially striking. He was a small, shrivelled, wizened child, with a pinched, aged expression. The ribs were distinctly beaded, but apart from this there were no evident signs of rickets. The hands were in the characteristic position of tetany, and facial irritability and Erb's and Trousseau's phenomena were easily elicited. The abdomen was generally distended, and by palpation gave the impression of being packed with contents, but nothing more definite could be made out on the first examination. His stools were found to correspond closely to those of the boy described under Case 1. They were large, whitish, pultaceous in appearance and very offensive. The effect of washing out the large intestine in the first case was so beneficial that he was treated in the same way. The first enema evacuated a large quantity of the characteristic fæcal material, and subsequently saline enemata up to the volume of two pints were easily retained for several hours. After he had been in the hospital for two or three days and his large intestine had in this way been emptied, signs of a large, hollow viscus could be detected by abdominal examination. By percussion a drum-like note could be produced over the epigastric and left hypochondriac, lumbar, and iliac regions, and in limited areas, notably in the left iliac region, a distinctly tubular note. On two or three occasions peristalsis was seen over the same area, but it was impossible to recognise its direction. A week later the stools had become yellow and had lost their characteristic consistence, the abdomen was less distended and more flaccid and the tetany was much less. It seemed necessary, if attacks of tetany were to be avoided in the future, to operate and remove the dilated gut, but he was far from a fit subject for any such procedure; indeed, it seemed unlikely that he would survive it. For this reason medical treatment was persisted in, in order to at first improve, if possible, his general condition. Careful dieting and daily lavage promised to be

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PLATE XI.

Illustrating Dr. Langmead's Case of Tetany associated with Dilatation of Large Intestine.

Photograph *post mortem*, showing the distended cæcum and ascending colon.

successful, and at first he gained weight and became generally better. The tetany did not return and the motions remained normal. Unfortunately, however, his improvement was only temporary, and on June 6, nearly a month after admission, he developed diarrhœa, refused his food, and began to lose the weight he had gained. On June 8 Mr. Arbuthnot Lane operated and removed about ten inches of dilated sigmoid flexure. No more manipulation was used than was absolutely necessary and no further examination was made. For a few days an improvement was evident, but afterwards the pultaceous motions recurred, five or seven daily, and death took place from exhaustion on the twelfth day after the operation.

When the abdomen was opened *post mortem* it was at once evident that the large intestine was considerably dilated throughout, and filled with gas. In the undistended state the appearance of the gut was striking enough, but on inflation its size was seen to be very remarkable. Two large loops of intestine were so produced (Pl. XI), one formed by the cæcum and first part of the ascending colon, the other by the transverse colon, the intervening part being also distended, but to a less extent. The two loops seemed to fill the abdominal cavity completely, hiding the rest of the contents from view. The distribution of the dilatation was due to the peritoneal attachment, since, when the colon was removed from the body, the dilatation became uniform. The large intestine measured twenty-five inches in length. Its walls were not hypertrophied, but thin and velamentous, such as are found in cases of starvation or cholera infantum. No obstruction was present. The small intestine was not dilated. The sigmoid flexure had been removed at the operation, and end-to-end anastomosis effected, resulting in a perfect junction. No other morbid change was found.

Case 3.—The account of this case I have found among the records of the Hospital for Sick Children, and it is the only example of this association among them. It is that of a girl, æt. 2½, who was admitted under the care of the late Dr. Sturges, on June 17, 1884. Her history was as follows: Up to the age of 14 months she had been quite healthy, but then developed diarrhœa, passing slimy, green, offensive stools, containing undigested curd. She began to waste rapidly, and in contrast to her general wasting her abdomen became distended. At the same time she became subject to

attacks of tetany, which lasted, according to the mother, sometimes for a week, sometimes for a shorter period. The attack of tetany for which she was admitted had been present for two days. In the ward she was seen to be an emaciated child, with a pinched, monkey-like face, whilst her abdomen was large, full, and resistant. Diarrhœa is noted as having been present, but no further description of the stools is given. Laryngismus developed if she became excited. Diarrhœa, tetany, and emaciation continued until she died from a terminal broncho-pneumonia a month later. *Post-mortem* the body was extremely emaciated, weighing only ten and a quarter pounds. The only lesion found except broncho-pneumonia was a dilated transverse colon.

A patient showing somewhat similar symptoms was exhibited before this Society in January of last year, by Dr. Batty Shaw and Mr. Mant. She was an ill-nourished child, and for four years had been subject to gastro-intestinal disorder. For eighteen months her abdomen had been distended, and for the same time she had been unable to walk because of weakness of the lower extremities. Repeated attacks of tetany had occurred for seven months. To her generally ill-nourished condition were added considerable secondary anæmia, puffiness of the face, and firm œdema of the feet and hands. Her knee-jerks were at one time lost. At first the tetany disappeared while she was being treated with salol and other antiseptic measures by the mouth, but when the condition recurred this treatment proved useless. Thyroid substance was given, and the tetany soon went. I am able to add another point to the clinical history of this case by the kindness of Dr Batty Shaw. Shortly afterwards the abdomen was explored by Mr. Bilton Pollard, and a general enlargement of the large intestine was found.

These four cases form a very definite group, which has apparently hitherto gone unrecognised; they have four common attributes:

(1) Obstinate relapsing tetany, (2) abdominal distension, (3) offensive, unhealthy motions, and (4) a dilated large intestine, as verified either *post mortem* or by laparotomy.

In Cases 2 and 4 this dilatation of large intestine was general, in Case 1 limited to the sigmoid flexure, in Case 3 to the transverse colon. Cases 1 and 2 were benefited when the dilated portions were emptied of their stagnating contents by saline enemata, Case 4 by the administration of thyroid extract.

The question which we may ask ourselves is, Does the study of this well-defined group of cases help us to understand more fully the cause of tetany generally? and to this I would answer that it is all in favour of a toxæmic origin of this condition. The dilated bowel filled with fermenting fæcal material, the continuation of the tetany until that is washed out or until thyroid is administered, the immediate improvement when this is done, and the return of the tetany with re-accumulation, all indicate clearly the absorption of some poison from the intestine—a poison perhaps normally absorbed in quantities capable of being neutralised by parathyroid secretion, but in these cases generated in too great abundance to be so neutralised.

In these four cases another factor was present, viz. prolonged malnutrition, and if we consider the recognised groups of tetany we find that either improper feeding or undue depletion is very frequently in evidence—*e. g.* in rickets it is the former; in gastro-intestinal disorder, gastric dilatation; and in lactation, the latter. This may well act by producing a hyperexcitable nervous system, such as is confessedly present in rickets, one readily stimulated by a circulating toxin. To argue from the particular to the general is notoriously unsafe, yet it must be conceded that the association of an unduly excitable nervous system with disturbance of the alimentary tract which is present in this particular form of tetany is present also in the great majority of the previously described cases, whether they were due to rickets, gastric dilatation, diarrhœa, lactation, or typhoid and other acute infection. Their treatment, then, like that of those which form the subject of this paper, would seem the treatment of the cause of malnutrition and then removal of the focus by operation or its contents by lavage, where these are practicable, or the administration of thyroid, or rather parathyroid extract, a substance which has been shown experimentally to antagonise the tetany-producing poison.

XVI.—*Case of Typhoid Fever, with Acute Pneumonia, Pericarditis, Endocarditis, and Hæmorrhagic Pleural Effusion; Recovery.* By Francis H. Hawkins, M.D. *Read February* 8, 1907.

ANNIE N., æt. 20, a worker in the biscuit factory, was admitted under my care on November 17, 1900, complaining of pains on the left side, in both hips, knees, and arms. She first felt ill on November 10, when she complained of sore throat and of stiffness in her legs. On November 15—that is, five days later—she had a shivering fit, which was followed by diarrhœa. There was no previous history of headache, epistaxis, or sickness. Until the present illness her health had been good. Her sister had been in the hospital with rheumatic fever, and both her father and mother had suffered from rheumatism.

State on admission.—Patient was a slender, fair-haired girl, somewhat anæmic, but well developed. There was no swelling of any joint, and no perceptible perspiration. The abdomen was but slightly distended, nor was the spleen enlarged. The tongue was moist and slightly coated. The bowels were moved twice shortly after admission.

Heart.—The apex-beat was in the fifth interspace in the left mammary line. The sounds were normal, but over the base of the heart was a well-marked systolic murmur. The pulse was 100; the temperature 103° F.

Further report of the case.—Vidal's reaction was given. No typical typhoid spots were at any time present. At times the patient complained of pains over the abdomen, which for some time remained considerably distended. The bowels during the first twelve days were moved once or twice a day, but from November 29 to December 18 they were moved from twice to seven times a day. The stools were of the typical pea-soup character. On December 4, the third week after admission, the patient complained of pain across the shoulders and over the epigastric region, and she developed a cough, together with blood-stained expectoration. The note over the left base was impaired, and the breathing was of bronchial character. Two days later (December 6) the expectoration was of a rusty type, and contained numerous pneumococci. There was now dulness at the left base, with well-marked

tubular breathing. Four days later (December 10) the tongue became dry and glazed, the cough was frequent, the patient became very cyanosed at times, and now slight pericardial friction was heard. For the next two days the patient seemed to improve, the tongue became moist, the cough lessened, and she slept better, but on December 19 her condition became critical, the lips and lobes of the ear became blue, and the whole face was cyanosed. She could not move, and when moved by the nurse the cyanosis increased. The cough was now constant, the respirations 42 per minute, the alæ nasi dilated in respiration, the pulse was 128, and a distinct apical systolic murmur was now heard. During inspiration the left side of the thorax was motionless; the dulness over the left base was absolute. The breath-sounds were markedly tubular, but somewhat distant; fine crepitations could be heard, and, while it was quite impossible to determine either the presence or absence of vocal fremitus, I felt sure that there must be effusion in the left pleural cavity, and therefore determined to perform paracentesis. It was done without any delay, and 14½ oz. of hæmorrhagic fluid were withdrawn. This gave great relief. The day following the cyanosis had disappeared, the breathing was less laboured, and the patient had slept well during the night. The cough was still troublesome, and pericardial friction much more marked. The pulmonary signs gradually cleared up, the pericardial friction had disappeared, but the systolic murmur at the apex still existed (and the second sound over the pulmonary area was accentuated) when the patient was discharged on January 24, 1901.

As to the temperature, you will note in the chart passed round that in the evening of the day of admission it was 103° F., that during the next two days it was 101·2° and 101° F. On the evening of the third day 99° F., that for the three following days it was normal or sub-normal, but on November 24 it was 100° F., and on the 26th it was 100·4° F. During this time the patient was taking salicylate of soda, which had been prescribed for her by the house-physician, he regarding the case as one of rheumatic fever. I had not myself been able to see the case till some days later, when, taking everything into consideration, I looked upon the case as one of typhoid, and the subsequent examination of the blood confirmed this opinion. On December 4, when the patient expectorated blood, the temperature at 3 o'clock in the afternoon was 103·2° F., but at seven o'clock the following morning it was 99° F. On December 6, the day of

the rusty expectoration, the temperature was 102° F. at 11 p.m., and remained between 100·2° and 102·4° F. until the 11th, when at 7 a.m. it was 99° F. This I regarded as the crisis although the temperature rose during the day, and at 7 p.m. was 101·4° F., not returning to the normal till the 14th, and perhaps this should be considered as the termination of the pneumonic fever, as it was not till the 14th that the pulse-rate, which during the six preceding days had been from 100 to 120, fell to 86.

Remarks.—I venture to bring this case before the Society because of the nature of the complications and the fact that the case recovered, and is now quite well, and has not been ill even for one day since her discharge from the hospital. While acute fibrinous pneumonia is by no means a rare complication of typhoid fever, it is not of common occurrence. Speaking, if I may be so allowed, of personal experience, the case now recorded is the only instance of such a complication with complete recovery that has come under my own observation during a period of twelve years' hospital work and in a series of over 100 cases. In 492 cases clinically considered in the *Middlesex Hospital Reports* "pneumonia" was stated as a complication in eleven cases; of these eleven cases five died. All of these were instances of acute fibrinous pneumonia; the right lung was affected in all cases. Fränkel, in 500 cases of typhoid clinically considered, found six cases of acute fibrinous pneumonia. Dr. Horton-Smith Hartley, in the *Goulstonian Lectures* of 1900, says that lobar pneumonia is a rather more frequent complication than is generally supposed, it having occurred "in nearly 5 per cent. of our fatal cases"—that is to say, that in an examination of all the fatal cases of typhoid fever in St. Bartholomew's Hospital during a period of thirty years, thirteen cases were complicated with acute fibrinous pneumonia.

Pericarditis, which was well marked in this case, is quite exceptional in typhoid fever. Curschmann, writing in Nothnägel's *Encyclopædia,* says that in isolated instances he had found fibrinous deposits upon the pericardium in association with complicating croupous pneumonia. Dr. Horton-Smith Hartley found pericarditis present in 0·68 per cent., or in two cases only in 290 autopsies. And now as to the significance of the systolic murmur heard at the apex in the case herein recorded. At the time I hesitated to pronounce it as evidence of endocarditis, knowing how difficult it is to distinguish an endocardial murmur from one due to typhoid

myocarditis, with relative mitral insufficiency, but the murmur being present some time after seemed to imply that it was consequent on endocarditis. An examination made a few days ago confirms this, for there is now a well-marked systolic murmur at the apex, propagated into the axilla, accompanying the first sound. I might here refer to the case of a fellow student, in Edinburgh, who contracted typhoid. During the course of the disease murmurs developed over the aortic area, and some fifteen years later he died from heart disease. Endocarditis, according to Curschmann, is rare in typhoid fever. In none of the 492 cases clinically considered, before referred to, was any cardiac murmur recorded, but in sixty-one necropsies of typhoid fever cases in the *Middlesex Hospital Reports* I find two instances recorded where the mitral valves were affected, in one case as "early mitral endocarditis," and in the other, "recent vegetations on mitral valve." According to the results of Dr. Horton-Smith Hartley's investigations endocarditis is rather more frequently met with than is pericarditis. He found it in 1·37 per cent., or in four fatal cases during a period of thirty years. As to pleurisy in typhoid, writers seem to differ as to its frequency, but all seem to agree that pleural effusions, to any extent, are not common; indeed, Curschmann considers pleural effusions, of any amount, to be remarkably rare. In the 492 cases, to which I have before referred to as being considered clinically, "pleurisy" was noted in four cases, and "pleural effusion" in two; in one of these two cases hæmoptysis occurred, and was thought to be caused by pulmonary thrombosis. Subsequently a pleural effusion complicated the case, but a good recovery finally took place. In sixty-one autopsies made at the Middlesex Hospital, and recorded in their *Reports*, the pleura was noted as being affected in six cases. In one case there was "early pleurisy," and in another "fibrinous exudation on pleural surface." In this case the lung was in the stage of grey hepatisation of acute pneumonia. Pleural effusion was present in two cases; in one case the pleural cavity contained 6 oz. of clear fluid, the lung was in the stage of grey hepatisation, and in another case 4 oz. of dirty fluid was in the pleural cavity; the lung in this case was "gangrenous," and in the third case the pleural cavity had been opened. In one case petechiæ were noted as being on the pleural surface.

Dr. Horton-Smith Hartley found dry "pleurisy" in sixteen cases, being primary in five and secondary to other lung

complications in eleven. He found "pleural effusion" in twelve cases, it being primary in nine, and secondary to other pulmonary complications in three.

Hæmorrhagic pleural effusion is rarely met with as a complication of typhoid fever; it is a very serious complication. I cannot find any case recorded where the patient recovered. Dr. Horton-Smith Hartley does not record a single instance as occurring in the fatal cases in St. Bartholomew's Hospital during a period of thirty years, but he found subpleural hæmorrhages in six cases. Louis found, in nineteen ont of forty-six fatal cases, a greater or less amount of reddish serous effusion in the pleural cavity. In most of these cases there was hypostatic consolidation of the lungs. Curschmann writes: "A number of hæmorrhagic effusions that I have encountered proved to be tuberculous." In the case now recorded there was no suspicion or evidence of any tuberculous disease, and no tubercle bacilli could be found in the fluid withdrawn.

XVII.—*Juvenile General Paralysis of the Insane.* By John R. Lunn, F.R.C.S., and Gordon Holmes, M.D. *Read February* 8, 1907.

CLINICAL and *post-mortem* notes of a case, shown at the clinical meeting of this Society, January 26, 1906, which was supposed to be a case of general paralysis in a boy, æt. 16.

The boy remained in the same condition until February 15, 1906, when he had an unconscious fit, which was accompanied by more weakness of his left arm and leg. In August, 1906, it was noted he had right facial paralysis and a spasmodic spastic condition of the left arm and leg with incontinence of urine. On September 15 he had another fit with conjugate deviation of his eyes to the left and increased knee-jerks. He died September 13, 1906.

Post-mortem notes.—The skull was opened from behind and the brain and cord removed. A recent blood-clot came away (about 8 oz.) and was apparently situated between the dura mater and arachnoid membrane and had compressed the brain considerably on the left side. No rupture of any sinus could be found; the meningeal vessels were intact. After the removal of the brain, blood could be squeezed out of the cerebral veins in two places, one over the left frontal lobe, the other over the left occipital. This was thought might possibly be the source of the hæmorrhage. No other lesion was found in the other viscera.

Dr. Gordon Holmes has kindly undertaken to describe to you the pathological changes that he found in the brain, and thinks with me that this case must have been one of general paralysis of the insane, which confirmed the diagnosis.

Pathological Examination.

Except for some flattening in the left parietal region, which was due to the large subdural hæmorrhage that was found at the autopsy, the brain appeared normal to the naked eye. There was no marked wasting of any part of it, and the meninges of both the surface of the hemispheres and of the base seemed natural. No ruptured vessel or tear in the pia mater of the left hemisphere could be found, so the subdural

hæmorrhage could not have come from the brain, but probably arose from the rupture of a vessel in the arachnoid or in the dura mater.

No disease could be seen in the brain when it was cut up, except that the walls of the lateral ventricles, and especially the floor of the fourth ventricle, were granular.

Practically the same changes were found in all parts of the brain on microscopical examination. The most striking change was the presence of cellular infiltration around the vessels into their adventitial sheaths and perivascular spaces. In suitably stained sections (with Heidenhain's iron-hæmatoxylin and Unna-Pappenheim's stain) the following types of cells could be easily recognised: (1) Small round nuclei with none, or very little protoplasm around them; these appear to be lymphocytes. (2) Similar nuclei surrounded by a considerable zone of protoplasm; these correspond to the plasma-cells of Marscholko. (3) Larger cells, in the protoplasm of which coarse granules are visible; these are mast-cells. (4) Cells more elongated or irregular in shape and with larger and paler nuclei; they are probably proliferated connective-tissue cells of the adventitial sheaths. Some proliferation of neuroglial nuclei could be seen in the neighbourhood of many of the vessels. Despite this great pathological alteration around the vessels, there was, as a rule, very little evidence of disease in their middle and inner coats; there was no proliferation of the endothelial cells, but a slight degree of hyaline degeneration of the walls of a small proportion of the vessels was seen. In places, chiefly in the depths of the sulci, the vessels of the pia-arachnoid were surrounded by collections of similar cells, and here and there the soft membranes were infiltrated by them, apparently without any relation to the vessels.

Besides this perivascular cell infiltration, numerous clumps of cells, more or less similar to those around the vessels, but containing oval or irregularly-shaped nuclei in large proportion, were found in all parts of the brain. They occupied irregular areas without definite limits, and there was surprisingly little evidence of reaction in the tissue around them, though some neuroglial proliferation could be generally detected.

These changes were on the whole more marked in the grey than in the white matter of the brain, and were especially pronounced in the cerebral cortex. They were very much less in the brain-stem, where practically the only

change was slight perivascular infiltration in the grey matter. The soft membranes of the cord were similarly affected, but it was only along the pial septa and the vessels which enter it from the pia mater that any cellular infiltration was seen within it.

In sections of the cerebral cortex stained by Nissl's method it was seen that the majority of the cells were affected, though only in slight degree. In practically all there was considerable chromatolysis—*i. e.* disintegration of the Nissl bodies, with or without excentricity of the nucleus. Other cells which were more severely affected appeared atrophied and shrunken and stained badly. A few were seen in which the affection was more acute, the cell being swollen and in the state of chromatolysis with its nucleus much enlarged.

The state of the nerve fibres was investigated by Weigert's method. The chief alteration found was great atrophy or complete absence of the horizontal layers of fibres of the cortex, especially of the tangential and supra-radial layers, and a partial degeneration of the pyramidal tracts, which could be followed from the internal capsule to the lower segments of the cord.

There was no visible disease in the larger vessels of the brain, and it was only in a few of the larger pial vessels of the cord that any endarteritis could be found. This was the only evidence of syphilis that was discovered in the central nervous system.

The pathological changes above described indicate the existence of a chronic inflammatory process, which may be most probably ascribed to the action of toxins which affected the central nervous system through the blood-vessels. These changes are identical with those which are regarded as pathognomonic of juvenile general paralysis of the insane. The pathological examination of this case, therefore, confirms the clinical diagnosis.

XVIII.—*A Case of Idiopathic Dilatation of the Rectum and of the Colon as far as the Hepatic Flexure.* By H. Morley Fletcher, M.D., and H. Betham Robinson, M.S. *Read February* 8, 1907.

At a meeting of this Society, held April 23, 1905, we exhibited the patient who presented the above lesions as a clinical case. The boy, who was under our care at the East London Hospital for Children, has since died, and we now bring before the Society a fuller account of the case, which showed some anatomical features unusually met with in this condition.

P. V., æt. 12, was admitted into the hospital on November 15, 1904. The mother stated that the boy had always had a "large belly" and been rather constipated, but not markedly so until within the last fortnight; otherwise he had been a healthy lad, though not robust. A fortnight before admission she noticed a great increase in the size of the belly, constipation became more marked, and the boy complained of some pain on defæcation; there was no vomiting, nor was there any passage of blood *per rectum*. This condition with some wasting persisted until admission.

On admission he appeared to be a nervous lad, very sallow, and with a very distended belly. His heart and lungs, pulse and temperature were normal. The abdomen was tense, with a regular rounded outline and no widening of the costal angle. The surface veins were rather full. The percussion note was resonant all over, and in the knee-elbow position a thrill was obtained over a small area round the navel. On palpation irregular masses were felt in the region of the transverse colon. After examination peristalsis was very obvious. On rectal examination the tone of the sphincter was normal, but the rectum was very dilated. The belly measured in circumference at the navel 29 in., from navel to pubes was 5¼ in., and from navel to ensiform 7¼ in. His weight was 3 st. 4 lb. 14 oz.

On November 25 the following note was made: There was a slight increase in girth, and the bowels were obstinately constipated; there was no response to aperient medicines and only poor results from enemata. The weight had increased 1 lb.

At no period was there tetany.

On November 29 Mr. Robinson operated. A mid-line incision was used below the umbilicus, and on opening the peritoneal cavity about two ounces of clear fluid escaped. A large, tense, bluish-coloured tumour presented with very large distended veins coursing over it, and with a lace-like pattern on its surface due to the hypertrophied and separated muscular bundles. The bladder was attached to the lower part in front. The swelling was proved to be free at the sides and in front, and to extend upwards in the mid-line to the ensiform cartilage. To further explore this the incision had to be extended considerably upwards and the tumour was brought out of the belly. The following was found to be the condition of things. The mid-line swelling started from the pelvic floor and expanded immediately like the body of a flask; above the navel it began to narrow into the neck of the flask, but yet its circumference was as large as a man's arm. Just below the ensiform cartilage there was a somewhat acute bend to the left, and the gut, with almost the same calibre, went straight down into the left iliac fossa, formed a loop and became continuous with the descending colon. Further exploration showed the dilatation continued along the descending and transverse colon with very little diminution in size until the hepatic flexure was reached. The ascending colon was only slightly enlarged and filled with some hard fæcal lumps, and the cæcum and appendix were normal. The colon beyond the hepatic flexure contained many pounds of soft, dark, fæcal material, which was squeezed out *per anum* by gradual pressure on the gut from within, with dilatation of the anal canal by the assistant's finger. It was remarked at the operation when this had been evacuated that the hypertrophy of the bowel wall was so great as to feel "as thick as the sole of a boot." The finger, after passing through the internal sphincter, entered an enormously dilated cavity whose walls were touched with difficulty. No hard or enlarged glands were felt in the mesentery; there were no signs of peritonitis, nor any abnormal band. The rectum, before emptying, was measured with an aseptic silk ligature at its greatest circumference from mesentery to mesentery, and proved to be $19\frac{3}{4}$ ins. The abdominal wall was closed in layers.

After the operation the boy did well except for slight suppuration in the superficial layers of the wound. He was put on a tonic mixture, and passed one to four motions daily without further medicine. On December 14 he had

again constipation, and he was put on aperients and the lower bowel washed out every day. On January 18 the belly was larger and hard lumps were felt; no motion was passed, in spite of strong purgatives, and enemata also gave no result. An enema, ol. ricini, on January 24, brought away nine actions, and after that the belly became soft, and a daily enema was effectual. After a few days these were discontinued and the boy had a daily motion; his weight increased and his abdomen was normal in size and softness.

During the following months the boy remained in excellent health; he steadily gained weight, and had a regular action of the bowels. He was seen at the hospital from time to time when he came to report himself.

On March 17, 1906, he was re-admitted under one of us (H. M. F.), and remained in the hospital for eleven days in order that his condition might be accurately observed. He appeared to be perfectly well, and except for the scar from the operation the abdomen seemed to be quite natural, and was free from any abnormal distension. The bowels acted regularly and naturally without medicine. Rectal examination showed that there was some ballooning of the gut and nothing else could be made out.

The boy continued in good health, except that lately the bowels had been confined, till the evening of July 2, when he came home from school complaining of abdominal pain. No distension was observed by his mother. The pain was relieved by ginger brandy, but shortly afterwards diarrhœa set in and continued till 6 a.m. the following morning. There was no pain during the night. The motions were dark, not offensive, and did not contain blood. No delirium during the night. Next morning, at 8 a.m., his mother started from home to bring him to the hospital. According to her account he was quite conscious then, though his breathing was very quick but not distressed. He was conscious when he arrived, but became comatose immediately on admission. There was no vomiting. He died in deep coma very shortly after he was admitted. There was no abdominal distension.

A *post-mortem* examination was made by Dr. Iredell. The body was well nourished, and there was no distension of the abdomen. The thoracic organs were natural. On opening the abdomen an enormously distended viscus presented—this was the rectum and colon. The appendix

was normal. A flat, narrow adhesion about a quarter of an inch wide, formed by a portion of the great omentum, was attached to the upper limit of the swelling, and this was the only adhesion present. The gut was dilated to a circumference about a quarter to a half greater than normal, and the dilatation began one inch below the splenic flexure. Both muscular coats were hypertrophied. There was a balloon-like dilatation filling up the whole of the pelvis and the greater part of the abdominal cavity, which was continuous with the sigmoid loop, as described at the operation. The wall was universally thickened and its longitudinal fibres were coarse and prominent, forming an interlacing network, most marked on the aspect furthest from the mesentery. A distinct mesentery, one inch in length, was present, uniting it to the posterior abdominal wall and allowing free mobility of the gut. In no place was there any marked contraction or any sign of thickening of the circular muscle coat. The intestines were filled with gas and liquid fæces. All the other abdominal viscera were natural.

I have brought here the whole of the large intestine from the cæcum to the external sphincter.

The anal canal is normal showing no unusual fold or stricture. At the upper level of the internal sphincter the bowel opens out immediately into the large cavity before spoken of. The greatest diameter of this cavity is seen in the rectum proper, which appears considerably lengthened, as well as dilated; the peritoneum remains still *in situ*, and indicates the region of bowel affected. The walls of the cavity are much thickened, both muscular layers appearing hypertrophied. The lining membrane seems quite healthy, with no signs of present or past ulceration; the longitudinal columns are reduced in size and separated, owing to the dilatation, but they normally converge at the pelvic floor. There is no abnormal fold, sacculus or constriction which might be a contributory cause of the dilatation. The rest of the bowel examined shows no further abnormality beyond what has been described as seen at the operation, namely general dilatation, but no obvious great hypertrophy of the walls.

The chief points of clinical interest in this case appear to us to be the following:

(1) *The absence of symptoms in early life.*—Except for the large size of the abdomen and a tendency to constipation,

nothing was noticed by the parents until about a fortnight before admission to the hospital, although we believe the condition to have existed from a very early date.

(2) *The suddenness of onset of symptoms.*—The rapid wasting with great abdominal distension accompanied by vomiting and obstinate constipation suggested an acute condition, and closely simulated tuberculous peritonitis.

(3) *The great improvement* in general health and the practically complete absence of the earlier symptoms which followed the operation. This, as we have stated, consisted only in completely emptying the gut, and was followed by careful dieting and regulation of the bowels.

(4) *Duration of the improvement.*—In many of the cases described by other writers improvement follows operation, but this has very rarely, if at all, lasted for so prolonged a period and has been so completely successful that the patient has been to all appearance in ordinary health.

(5) *The sudden termination with coma.*—The terminal enteritis may have been due to the ingestion of some toxic substance taken in by the mouth, such as tinned meat, or it may have been infective in origin, but the question arises, was its severity due to the abnormal condition of the gut with its greatly increased absorbing surface? As far as we can gather from published cases this form of termination would appear to be most unusual, if not unique.

As to the pathology of the condition, we can add nothing further. We would agree to the division into two groups, the one due to mechanical obstruction, the other probably congenital in origin in which no obstruction can be found. In the absence of any kinking, bands, sacculi, or diverticula, diaphragms, or other conditions which have been described as causing obstruction, we must include the case in the second group known by the unsatisfactory term of "congenital or idiopathic dilatation of the colon."

As to the surgical procedure adopted in this case, it may be said that it stopped at the first stage because of the satisfactory result produced. What we had decided to do if further treatment was necessary was to open the cæcum so as to wash out and give complete rest for a time to the bowel beyond. Should this not have produced the required contraction, the question then to be considered was the performance either of some form of anastomosis or of excision of the dilated bowel. Both these procedures have been adopted, but this case, unlike any other previously described, presented

conditions which would have made either of them a very doubtful remedy. As to the resection, it would have been necessary to remove the dilated rectum and the mucous membrane of the anal canal, following the plan adopted by Treves, and to place here healthy small intestine which would have been the ascending colon, or the terminal part of the small intestine. In both such considerable difficulty and risk would be encountered in attempting these measures as to almost prohibit its trial. If an anastomosis had been attempted it would have been between the ileum and the large rectal dilatation. This might have had some success provided the dilated bowel had been thoroughly emptied of its contents at the time of making the anastomosis, but then, again, there would have been a possibility that the intestinal contents might have accumulated in the dilated rectum and made matters much as before. To overcome this liability to accumulation, plication of the redundant rectal walls might have been performed.

From the degree of success of our case we would urge that a similar procedure of emptying the bowel by abdominal section be attempted as a routine method before proceeding to more serious operations. The *post-mortem* appearances of the bowel showed that considerable diminution in the calibre of the intestine had taken place, and we know that this improvement had been accompanied by loss of his previous symptoms —constipation, distension and wasting. We assume that, but for his somewhat accidental death, he might possibly have completely recovered. In most of the cases previously recorded there was ulceration of the bowel, and the fact that in our case the bowel showed no ulceration but a healthy condition of the mucous membrane may have contributed to the favourable result.

XIX.—*A Case of Spinal Meningocele in an Adult successfully treated by operation after Spontaneous Rupture had occurred.* By Raymond Johnson and C. E. Reynolds. *Read February* 22, 1907. *With Figures.*

THE patient was a healthy, well-developed woman, æt. 22. Her mother stated that since birth a small depression had been noticed at the lower part of the spine. In June, 1903—the patient being then nearly twenty years of age—it was first observed that a swelling was present immediately above the depression; the swelling gradually increased in size. The swelling was painless, but if it was knocked the patient experienced the sensation of a "quiver" passing up to her head. On December 29, 1905, the patient noticed that her clothing was wet, and it was found that fluid was leaking in considerable amount from a small opening which had formed on the surface of the swelling. On the following day she was sent up to London from Somersetshire, where she lived, and was at once admitted into University College Hospital.

The condition on admission was as follows: The general state of the patient was normal. Over the lower part of the sacrum, in the middle line, was a hemispherical, sessile swelling, three inches in diameter. Over the greater part of its surface the tumour was covered with rather thin, but otherwise normal, skin. On the summit of the swelling, however, was an area nearly as large as a sixpence, in which the skin was replaced by a thin translucent membrane, and in the centre of this area was a minute opening from which clear cerebro-spinal fluid escaped in large quantities. During the act of coughing the tumour became much more tense and the escape of fluid more copious. Immediately below the tumour, and in a position corresponding to the base of the coccyx, the skin presented a small, blind, conical recess, containing some sebaceous matter; this recess was overhung by the lower edge of the tumour. Beneath the tumour it could clearly be felt that the posterior wall of the sacral canal was deficient, the edges of the defect being traceable downwards on either side of the depression in the skin above described.

The skin having been repeatedly cleaned and a large absorbent dressing changed frequently, operation was performed on the following day (December 31). The meningo-

cele was opened by the excision of an elliptical piece of its wall and the skin covering it, including the thin membranous area in which the opening was situated. The sac, which would have held an ordinary-sized orange, was lined with a smooth glistening membrane. At its deepest part was a round opening, about 6 mm. in diameter, which led into the theca; from this opening the fluid in the sac alternately welled out and was sucked back during expiration and

inspiration. A circular incision was next made around the deep part of the sac at a distance of about 3 cm. from the deep orifice; the part of the sac thus included was then reflected to the edges of the opening and the latter closed after the manner of a hernial sac with two separate silk ligatures. The remainder of the sac was then removed and the skin wound sutured, the skin being depressed into the cavity left by the removal of the sac. A large dressing of cyanide gauze was applied. The patient was kept in the

prone position and the foot of the bed raised. No symptoms referable to the nervous system occurred at any time after the operation. During the first four days the maximum temperature was 100° F.; on the fifth day a further rise occurred and was maintained until the ninth day, when the maximum of 102·8° F. was reached; a rapid fall then occurred, and on and after the eleventh day the temperature was almost constantly normal. At the first and second dressings on the second and fifth days there was no evidence of any leakage of cerebro-spinal fluid, but on the sixth day there was a rather free blood-stained discharge. From this date onwards the large gauze dressing was changed daily and a free escape of cerebro-spinal fluid occurred from a small opening in the middle of the scar. The fluid was perfectly clear and there were no signs of inflammation, but on the ninth day, when the temperature reached its maximum of 102·8° F., a pure culture of *Staphylococcus albus* was obtained from the fluid discharge. On February 6—thirty-seven days after the first operation—a further operation was performed. The scar was opened up, and the small aperture in it was found to lead down to an opening into the theca, from which cerebro-spinal fluid still escaped. The opening was so small that it could be completely closed by clamping it with a pair of Spencer Wells' forceps. Whilst the opening was thus clamped a fine silk suture was passed continuously backwards and forwards through it with a curved needle and the ends tied. No further escape of fluid occurred, and the closure was made more secure by a second suture applied continuously to the more superficial fatty tissue. Finally the skin incision was completely closed. No further escape of cerebro-spinal fluid took place and primary union occurred in the wound. The patient left the hospital on March 9, and several months later was reported to be in perfect health.

Spinal meningocele is said to be more common in the sacral than in the other regions of the spine, the next in frequency being the cervical region, although, as Ballantyne points out, it is probable that in some of the cases recorded as cervical meningoceles the tumour was, in reality, occipital, and not cervical in origin. It is rare in the dorsal and not frequent in the lumbo-sacral region, where, as is well known, meningo-myelocele, the commonest form of spina bifida, usually occurs.

It is a striking feature of the case under observation that the tumour was not noticed until the age of twenty

years, although a well-marked coccygeal dimple had been present from birth, and that within two and a half years the cyst had undergone spontaneous rupture. It can hardly be supposed that the meningocele dated its actual origin from this period, and must be concluded that a small protrusion of the spinal meninges, causing at first no projecting tumour, underwent gradual enlargement. We are, however, unable to suggest any explanation for this occurrence. It is a further point of interest that during the period of more than a month, when cerebro-spinal fluid was constantly escaping in large quantities, the patient presented no symptoms referable to the loss. On three occasions only did any definite elevation of temperature occur. Two of these, accompanied with headache, coincided with the menstrual periods, the maximum temperature being 100° F. and 101·6° F. The third elevation of temperature, occurring on the ninth day after the first operation, reached a maximum of 102·8° F., and was coincident with the presence of the *Staphylococcus albus* in the fluid discharged. It is noteworthy that this infection was not accompanied with any local signs of inflammation, except that the fluid which escaped at this time was, perhaps, not quite so absolutely clear as usual. This observation is in keeping with the recent observations of Dudgeon and Sargent, who find that the *Staphylococcus albus* can, not infrequently, be demonstrated in wounds, pursuing apparently a perfectly aseptic course.

XX.—*Carcinoma of Jejunum; Obstruction; Resection of Jejunum and Involved Colon.* By C. H. Fagge, M.S. *Exhibited January* 25, 1907. *Read February* 22, 1907.

KATHERINE H., æt. 33, a married woman, was admitted into Guy's Hospital on December 11, 1905, for severe abdominal pain with vomiting and a lump in the right iliac fossa. Patient first noticed abdominal pain three months ago; during the attack she was sick. The pain and sickness recurred every three or four days, but for three weeks the pain has been more constant. She is unable to keep any food down and has lost flesh. During the attacks of pain the lump becomes larger.

On admission patient looks ill; pulse 118; temperature 96° F.; vomit is brown in colour. There is a hard, defined movable lump, the size of the palm, in the cæcal region. To the left of, and passing into, this lump can be seen, at times, a distended intestinal coil about three inches across; the peristaltic wave passes from below and to the left of the umbilicus towards the mass on the right. As the pulse remained at 120, and patient vomited several times, operation was undertaken about four hours after admission.

An incision in the right semilunar line exposed distended small intestine, which, on the right, ended in a hard mass of growth and was adherent above to the under surface of the transverse colon, near the hepatic flexure. The dilated, hypertrophied jejunum, some eighteen inches from the duodeno-jejunal flexure, was narrowed in two places by a mass of annular growth, and nearly the whole circumference of the colon was welded with the jejunum into a hard mass; there was much lymph, both around the bowel and on the mesentery. An attempt was made to separate the colon from the jejunum, but the mucous membrane of the latter was exposed. At this stage I thought I was dealing with a primary colic growth involving the jejunum secondarily. The jejunum was double ligatured both above and below the growth, and its proximal end above and its distal end below were invaginated and closed; the colon was treated in the same way, the part eliminated extending from the middle of the ascending to the middle of the transverse colon. The meso-colon, omentum, and mesentery were now ligatured and

the mass was removed: several enlarged, hard glands were felt in the mesentery. A lateral anastomosis, with a small Murphy's button, was now carried out between the two cut ends of the jejunum, and the colon was similarly restored by a large Murphy's button. The abdomen was washed out with saline solution and closed with a single layer of salmon-gut sutures. The operation lasted one and a half hours, and the patient, who was much collapsed, was improved by an injection of strychnine and continuous infusion with normal saline.

The sickness soon ceased, and her bowels were opened with enemata on the second day and by a purgative on the fifth day after operation. The large Murphy's button was passed at the end of four weeks, after a dose of castor oil, and the smaller button appeared two weeks later (January 17, 1906). Patient left the hospital on January 20.

The specimen, shown at the previous meeting, consists of about eight inches of the jejunum, beginning some fifteen inches from the duodeno-jejunal flexure; at its centre is an ulcerated area extending for about three inches, which, before it was laid open, produced two distinct strictures of the bowel; the higher stricture is the thicker and the narrower; the bowel above the specimen was seen to be much hypertrophied and dilated, and this, no doubt, was the coil in which the peristaltic wave was visible before the operation.

The growth around the second stricture has extended above to the peritoneal surface of the gut, and here involves the lower aspect of the hepatic flexure for some distance, though it has not reached the mucous membrane. The adherent mesentery contains several enlarged and hard glands.

Microscopical sections of a piece of the upper ulcer show much necrosis and few cancer cells scattered about in the muscular layers of the bowel, but a second section shows a typical spheroidal-celled carcinoma, which, preceded by a small-celled infiltration, involves the circular muscle coat. This section was referred to Mr. Targett, who reported: "this is a carcinoma of the scirrhous type such as is often met with in the stomach." Sections of the mesenteric lymph glands show no secondary deposits, and their induration is therefore probably inflammatory.

Remarks.

Consideration of this case may be approached from two standpoints: its interest as a case of acute obstruction in

which resection was imperative for physiological reasons, or as a case of primary carcinomatous growth of the small intestine. I propose to approach it from the latter aspect. In Bland-Sutton's *Tumours*, 1906, the author says: "As far as I am aware the diagnosis of primary cancer of the small intestine has not been made, for when seated in the small gut below the duodenum cancer usually gives rise to symptoms of acute obstruction." While malignant connective-tissue tumours of the small intestine are relatively common, malignant epithelial growths are rare. It would be an interesting digression to theorise upon the reason why carcinomata are so rare in the small intestine while so common in the stomach and colon.

This variety becomes greater still if we exclude those cases of carcinomata of the ileo-cæcal valve, which may have arisen in the cæcum. Barker, in the *Transactions* of this Society, vol. xxxiii, records an interesting example of excision of the cæcum for a cancer of the ileo-cæcal valve, and reviews other cases of growth in the same position.

Probably the commonest seat of primary carcinoma in the small bowel is at the ampulla of Vater in the duodenum. De Havilland Hall (*Lancet*, 1902) and Rolleston (*Lancet*, 1901) have recorded cases, and the latter, among some twenty cases of carcinoma involving the ampulla, thinks that seven may be regarded as true cases of primary cancer of the ampulla, the remainder probably arising in the bile-duct or pancreas.

In searching through the books of reference available at the Royal College of Surgeons' Library I have found only two cases which cannot be regarded as doubtful. The first is a case of multiple carcinomata of the ileum, and recorded by Bunting in the *Johns Hopkins Hospital Bulletin* for 1904.

Halsey and Peterson (*Postgraduate*, N. Y., 1905, vol. xx, p. 401), record the second, which is that of a male, æt. 46, whose obstruction was due to a growth in the jejunum, eighteen inches from the duodeno-jejunal flexure; the obstruction was overcome by short-circuiting the small intestine; the patient died a few months later, October, 1904. A gland removed from the mesentery showed "deposits of adeno-carcinoma of the scirrhous variety.

In the catalogue of the Gordon Museum, Guy's Hospital, there are records of three cases of primary carcinoma of the small intestine—two spheroidal-celled carcinomata in the ileum and one columnar-celled carcinoma in the jejunum; none of them were subjected to operation.

In the Pathological Society's *Transactions*, vol. xliv, Dr. Voelcker reports a case of carcinoma of the jejunum, adherent to and involving the ascending colon, and in the same volume Mr. C. A. Morton records a case in which the small intestine was opened and drained for a columnar-celled carcinoma of the lower end of the ileum, with an account of the subsequent autopsy. Multiple carcinomatous ulcerations occurring in the small intestine seem usually to be secondary, as in a specimen in the Guy's Museum where three constrictions are seen due to secondary spheroidal-celled deposits in the serous coat. At the autopsy there was a spheroidal-celled carcinoma in the right ovary, which was considered to be the primary growth.

With regard to the surgical aspect of this case I shall do no more than ask for opinions as to the best line of treatment of growths involving both large and small intestines and producing acute obstruction. Should we under these circumstances undertake what must be extensive resections, which is a practice contrary to generally-accepted surgical principles in such cases?

XXI.—*Excision of Joints : a New Method of Operating at the Shoulder.* By Thomas H. Kellock, B.C.

Read February 22, 1907.

EXCISION of joints for disease, or after injury, is an operation of considerable antiquity, and one which has undergone comparatively little change or modification in recent years. Indications for its performance, if not quite as frequent as formerly, still arise, and with one or two exceptions it is practised in much the same way now as in the pre-antiseptic days. With the means at our disposal it is now often possible to render the parts to be operated on quite aseptic, and so the necessity for subsequent amputation less frequent, and it is now also made possible to secure fixation of the bones by artificial means in those cases where this is desired; but the principles of the operations, the incisions, the manner in which the bones entering into the formation of the joint are dealt with and the subsequent treatment remain much as before.

Under the term "excision of joints," however, there are included operations of different natures performed in different ways and with different aims: at the knee, ankle, and joints of the tarsus, parts which have to bear the weight of the body, the object generally aimed at is stability and a firm synostosis of the ends of the bones—a real excision or total removal of the joint in fact, and this is brought about by the removal of as little as is possible from the ends of the bones, and the obtaining of bony union between the opposing surfaces by fixation either externally by means of splints or more directly by the use of some mechanical contrivance, such as wire, pegs, or screws, with subsequent complete rest for the part to aid in the union. In the upper limb, where free movement is of more importance than strength in the ultimate result, a different operation is described as "excision"; at the elbow and wrist, in order to obtain free movement fairly large pieces are removed from the bones on both sides of the joint, and early passive movements employed so as to bring about a false joint by wide longitudinal separation of the cut ends of the bones and absence of rest.

At the hip, shoulder, and temporo-maxillary joints the term "excision" is applied to a different operation again to what is in fact a partial excision : at these joints a very movable bone articulates with what is, relatively speaking, a fixed one,

and excision is generally taken to mean removal of more or less of the end of the movable bone with the aim—if, perhaps, the hip under certain circumstances be excepted—of bringing about a movable joint formed by the shortened bone on the one side and the articular surface of the more fixed bone on the other. The hip- and shoulder-joints are in many respects very alike, and yet the operation of excision as performed on them differs considerably, for at the hip the head of the femur is generally removed by section through the neck of the bone, that is, inside the capsule of the joint proximal to the insertion of the muscles into the great trochanter, and the part of the bone that is removed is free of muscular attachments. At the shoulder, on the other hand, with the object of obtaining a false joint that shall be freely movable, the head of the humerus is removed by section through the surgical neck, that is, outside the joint and with the head of the bone are sacrificed the insertions of the subscapularis, supra-spinatus, infra-spinatus and teres minor muscles, muscles which, if they could be saved, would be of very considerable use at such a joint as the shoulder, where a free and rapid use of the arm is of so much more importance than actual strength. It is true that after excision as it is generally performed the divided ends of these muscles are supposed to form fresh attachments, but it is doubtful if these can be to the shaft of the humerus, for their retraction after division and the large piece of the humerus that is removed must cause a large interval between their cut ends and the bone.

It occurred to me that when operating on the shoulder, in cases where the head of the humerus was not so extensively diseased as to render its removal imperative, it might be possible to remove the necessary bone from the scapular side of the joint instead of from the humeral, and by so doing save the attachments of these important muscles. Anatomically, the operation can be performed in this way with the sacrifice only of the long tendon of the biceps and a part of the middle head of the triceps, structures which are not of very great importance on account of the other origin of these muscles being retained. Even in cases of moderate disease of the humerus this method might still be applicable, for it must be remembered that in most cases disease affects both the head of the humerus and also the glenoid cavity. The general plan is to remove the head of the humerus at the surgical neck and scape or gouge away the affected bone from

the glenoid, and there seems to be no valid reason why these processes should not be reversed, unless, of course, the disease should have spread so far into the humerus as to render it necessary to remove this below the insertions of the muscles mentioned above.

The performance of the operation on the dead body shows it to be one of little difficulty; by making a rather long incision from the tip of the coracoid process downwards, along the anterior border of the deltoid muscle, the front of the joint is easily reached, and the tendon of the biceps, as it lies in its groove on the humerus, can be divided as its origin must eventually be sacrificed. The capsule of the joint having been laid open, the subscapularis muscle can be avoided by elevating the arm, but the coraco-brachialis and short head of the biceps coming from the coracoid process are considerably in the way, and the remainder of the operation is very much simplified if this process be broken through with bone forceps and turned a little downwards without completely separating it. I used an Adams osteotomy saw to divide the neck of the scapula, with some assistance to retract the soft parts; this is not a very difficult matter, although the thickness of bone to be divided is rather greater than one would have supposed.

A little while ago I had the opportunity of performing this operation on the living. The patient was a man, æt. 41, a blacksmith by occupation, the subject of an unreduced sub-coracoid dislocation, the result of an accident eleven weeks previously; at the time of the accident he also sustained a fracture of the surgical neck of the humerus, but this had firmly united in a fairly good position. He was not, altogether, a very favourable subject for a major operation as he had suffered from fits, probably of an epileptic nature, and gangrene of one lung, but at the time of operation he appeared to be in fair general health, and it was obvious something must be done for the shoulder, as the hand and arm were œdematous and comparatively useless, and he suffered a great deal of pain in them from pressure of the displaced bone on the cords of the brachial plexus.

The operation was performed through the same incision as on the dead body, but the pathological conditions made the approach to the neck of the scapula more difficult. The neck of the humerus was thickened as the result of the fracture, and the head, which was lying just under the coracoid process, was surrounded by a good deal of new bone, such as is generally found in this condition. By the use of an elevator

and movement of the limb the head of the bone was freed; the tendon of the biceps was then divided and the coracoid process broken with bone forceps and turned downwards. It was then found that the presence of the head of the humerus prevented the free use of the saw in dividing the neck of the scapula, so resort was had to gouge forceps with which the whole of the glenoid cavity and a portion of the neck of the scapula were removed. This took rather longer than when done with a saw, but I rather think it is the better method as one can nibble away the bone into something of a concavity. When this bone had been removed the head of the humerus fell back easily into the cavity and the coracoid process was then refixed by two sutures passed through the soft parts. Hæmorrhage throughout was insignificant and easily controlled. Silk sutures were used to bring the soft parts together; the skin wound completely closed without drainage and the arm and shoulder fixed in plaster-of-Paris. Primary union occurred throughout in a satisfactory manner, and after about ten days we began passive movements of the limb, the pain and swelling of the arm having disappeared immediately after the operation.

The result now, nearly three months after the operation, is very fairly satisfactory, and I hope considerably more improvement has yet to come; flexion and extension are quite good, abduction fair, but rotation of the humerus is poor.

Should the result in this case eventually prove satisfactory and occasion arise to perform the operation again, I think it might perhaps be easier in a case of unreduced dislocation to make a posterior incision as well as the anterior one so as to divide the neck of the scapula from behind, and I think I should also remove a little more bone than I did in this case.

Theoretically this operation should be particularly useful in the case of children, for in them, in addition to saving the attachment of the muscles, it would retain the epiphysial line and so cause much less shortening of the arm, as this is the end of the bone at which the greater part of its growth in length occurs, that is, of course, if the epiphysis were not already ruined as far as growth was concerned by tuberculous disease, which is by far the most common affection that calls for surgical treatment at this joint in young people.

XXII.—*The Medical Treatment of Congenital Pyloric Stenosis.* By G. A. Sutherland, M.D. *Read March* 8, 1907.

THE symptoms and diagnosis of congenital hypertrophic stenosis are now well known, and have been very fully described by Thomson, Cautley, Still, and others. The exact pathology of the affection is still unsettled, and it may be that the results of treatment will throw fresh light on the subject. This paper deals only with the medical treatment by means of dieting, and gastric lavage, a method of treatment to which Still has specially directed attention.* I propose to give brief notes of the last three cases which have come under my care, and then to make some observations on the treatment generally.

Case 1.—A male infant, æt. 6 weeks, a first child, was sent to me on May 22, 1906. The history was that the child had appeared to be healthy at birth, but that vomiting occurred during the first week, and had continued ever since. The vomiting was intermittent, sometimes only once a day, sometimes oftener, and usually occurred about twenty minutes after a feed. The vomited material was sometimes in large quantity, and was violently pumped out. The child was losing weight steadily; the bowels were very constipated, and acted only after the use of a glycerine suppository. The motions were small but healthy in appearance. The child was subject to flatulence, attacks of abdominal pain, and screaming attacks. It was always hungry and always ready for a feed.

The diet had been the mother's milk, and occasionally Allenbury's food and peptonised milk had been tried and been rejected. The breast milk had been examined and found to be satisfactory, although the chief constituents were rather small in amount (proteids 1·75 per cent., fat 2·4 per cent., sugar 5·06 per cent.).

The patient was a wasted, whining, unhappy-looking infant weighing 7 lb. 6 oz. On examination of the abdomen the stomach was seen to be contracting strongly, and peristaltic waves passed from the left to the right side of the abdomen. The pylorus was not felt. (Probably I do not hunt for the

* *Lancet*, March 11, 1905.

pylorus with the same care as others, as it does not appear necessary to do so, and the proceeding is very uncomfortable for the infant.)

The same night general convulsions supervened and lasted intermittently for thirty-six hours, but passed off under the use of bromide and chloral injections. The treatment ordered for the pyloric trouble was lavage once a day and a continuance of the breast feeding every two hours by day and every four at night, ten minutes at a time. On washing out the stomach a considerable amount of soft flocculent curd was removed, but this seemed to be of such a fine character that gastric digestion was evidently good. It was also considered that such a residue should pass easily through the pylorus unless abnormal spasm was present. The child remained under observation for three weeks, during which the progress of the case was as follows: The lavage was carried out every day for the most part, sometimes every second day. The amount of residual food left in the stomach was found steadily to diminish. The vomiting became much less frequent and less violent, and towards the end of the time was trifling. The bowels came to act naturally, without injections, and at times diarrhœa with green stools occurred. The employment of malt and oil, half a drachm thrice daily, seemed to produce gastro-intestinal disturbance. The child became much more comfortable and brighter with the cessation of the abdominal pain and vomiting. The weight at the end of three weeks was exactly the same as at the beginning.

The infant then passed from my care and was treated on the same lines by a medical friend for a few weeks, when the parents transferred the child to a surgeon as we did not advise operation. Apparently, however, he had not the courage of his convictions, for he temporised, and finally medical treatment by lavage was persisted with. Four and a half months later the following report was very kindly sent me by the doctor under whose care the child had been placed. Steady improvement had taken place, interrupted only by a series of convulsions, which the doctor had traced to the fact that the mother was menstruating at the time. The gain in weight was very slow but the infant now weighed ten pounds, *i. e.* almost three pounds heavier than when first seen. The weaning process was being carried out as the mother's milk was failing. The visible peristalsis of the stomach had passed off entirely two months previously.

Case 2.—A male infant, æt. 3 months, was sent to me on October 12, 1906. The child was healthy at birth, weighing 7½ lb., and was fed on cow's milk and water, which seemed to agree with it. At the age of five weeks vomiting commenced and had persisted. It occurred once or more daily, and was frequently very violent, the food being pumped out in large quantity. The bowels had been rather loose at first, but later constipation had been marked, and for the previous month the bowels had acted only after a glycerine suppository. The motions were extremely small. The child slept badly, had frequent attacks of abdominal pain, and was always ravenously hungry. There had been a steady loss of weight going on for about two months.

Many diets had been tried, including cow's milk and water, Mellin's food, peptonised milk, lacto, whey, and Allenburys' food, but none of these had had more than a temporary effect on the vomiting. The child had been fed quite regularly every two and a half hours by day, and somewhat less frequently by night.

On examination the infant was found to be extremely emaciated, restless, whining, and looking the picture of misery. The weight was then 6 lb. 13 oz. The temperature was normal. There was no sign of disease anywhere apart from the stomach. The abdomen was somewhat distended, and gastric peristalsis was elicited by rubbing the abdominal wall. The contraction waves were very strong, and passed right across from under the ribs on the left side to be lost on the right side of the abdomen. They were not apparently painful. The pylorus was not felt by me, but my friend, Dr. Cautley, who kindly saw the case with me, was able to palpate it.

As the infant was at the time on Allenburys' food that diet was continued, and washing out of the stomach was performed daily. The vomiting rapidly diminished, but complete digestion, as learned by the results of washing out, was not obtained until the feeding was employed every hour by day and every two hours by night. The bowels soon began to act naturally, and a steadily increasing amount of fæcal matter was present in the stools. The gain in weight was at first very slow, and some weeks showed a loss. The diet was varied with whey, peptonised milk, and cream, and a small quantity of malt extract, raw meat juice, and grape juice was given. The improvement was steady if slow, and there was a definite change for the better in the infant's comfort and

general appearance. The progress was interfered with at times by attacks of diarrhœa, and later by an attack of influenza with bronchitis. He passed out of my care at the end of three months, and was then a smiling, active infant, keenly interested in all that went on, and weighing 9 lb. 6 oz.—a gain of two and half pounds. The gastric lavage was being carried out every second day, as the influenza had left the infant very liable to gastric disturbance. Had it not been for this complication the washing out would probably not have been required so frequently. A further gain in weight of 1 lb. 5 oz. has taken place during the last six weeks.

Case 3.—A male infant, æt. 3 months, was admitted to hospital on April 20, 1906, suffering from marasmus. The child had been healthy at birth, but vomiting had come on a fortnight later and had continued. The diet had consisted entirely of the breast milk, but wasting had been persistent. The bowels were constipated, acting only every second day, and there had never been diarrhœa. There was nothing of importance in the family history and the patient had presented no signs of constitutional disease.

On admission the infant was found to be a typical example of "marasmus," very wasted, whining, always hungry and sucking its fist. The rectal temperature, night and morning, was 96° F. The weight was 6 lb. On physical examination the abdomen was rather distended and tense, and an inguinal hernia was present, but there were no signs of organic disease. A diet of equal parts of cow's milk, barley-water, and lime-water, half an ounce of each, was ordered; the feeding to be every two hours by day (sixteen hours), and every four hours by night (eight hours). At the end of twenty-four hours the quantity at a feed was raised to three ounces. After three days half a drachm of cream was given thrice daily. Under this treatment the child seemed to improve, gained six ounces in weight in ten days, and seemed more comfortable. The amount of cream was then increased to half a drachm at each feed, and this was quickly followed by vomiting. On returning to the former amount of cream the vomiting ceased. On May 9 it was noted that although the bowels only acted once a day and enemata were sometimes required the motions were satisfactory. The infant, however, was losing weight, although without any obvious discomfort or vomiting. Three ounce feeds of milk and lime-water, equal parts, every two hours by day and every four by night,

with half a drachm of cream and fifteen drops of cod-liver oil, thrice daily, were ordered. Vomiting quickly followed and continued. On May 14 examination of the abdomen showed marked peristalsis of the stomach, in waves passing from left to right, and a diagnosis of congenital hypertrophy of the pylorus was made. Washing out the stomach daily was ordered. The food was now changed to peptonised milk and water, and later cream, whey, plasmon, and plain milk and water were given at times. The amounts varied from three to four ounces at intervals of three or four hours. Although the stomach capacity, as determined on washing out, was about half a pint, I believe now that the feeds should have been smaller and less frequent. The washing out was continued daily, and there soon came to be but a small residue present in the stomach two hours after a feed. When, however, it was reduced to every second day, a considerable amount of food was found, and the daily routine was resumed. The vomiting was entirely relieved and occurred very rarely and in a very mild form. At the end of three months' treatment the weight was 6 lb. 11 oz.—a gain of only eleven ounces. The peristalsis of the stomach after a feed was as marked as ever. The temperature ranged between 96° and 97° F. Treatment by opium had been given a full trial, up to seven minims of liquor opii sedativus daily, without the slightest effect. After consultation operative treatment was decided on.

The operation of splitting the pylorus was performed by one of my colleagues on July 28. A typical large, hard, contracted pylorus was found. The only point about the operation which calls for notice was that the peritoneum was ruptured at the duodenal end of the pylorus, and was sutured. The infant suffered from a good deal of shock during the following few hours, but improved after saline infusions. Rectal feeding was employed for twelve hours and then one drachm of whey in an ounce of hot water was given every three hours. The amount of whey was steadily increased. On August 1 it was noted that there had been no vomiting for three days, the child was taking well and seemed comfortable. On August 3 the child did not take its food so well, and died suddenly and unexpectedly the same night. At the autopsy the abdominal conditions were healthy, there was no peritonitis, and there was no apparent cause of death.

Sudden and unexplained death in marasmic infants after a serious operation is, of course, of common occurrence. The

question as to whether better methods of feeding and other food materials might not have led to a cure of the pyloric condition without operation must remain unsettled, but further experience has led me to believe that the treatment might have been improved on.

Remarks on Treatment.

I shall not refer to the surgical treatment of this affection, a subject which was very thoroughly considered at the Toronto Meeting of the British Medical Association.* My hope and belief are that as our knowledge and experience of the medical treatment advance, there will be fewer and fewer cases in which surgical intervention is called for.

(1) *The objects aimed at.*—The aim of the medical treatment is to remove any source of irritation in the stomach, which may maintain pyloric spasm, and to keep the stomach free from any irritating food material, digested or undigested, which may excite pyloric spasm. Within recent years many cases of this affection have been seen in the *post-mortem* room, and as we looked at the hard hypertrophied pylorus and the complete stenosis it was a common remark that only operative treatment could have relieved the condition. It must be remembered, however, that the pylorus has two functions, contraction and relaxation, and the fact that the muscular tissues generally are hypertrophied is no reason for assuming that only the power of contraction is retained. I believe that both these functions are present at birth; and that there is, in addition, a congenital hyperplasia of the muscular tissues of the pylorus, as Cautley has so strongly insisted on. For a varying length of time—days or weeks in different cases—all goes well, and the pylorus acts normally. But a time comes, even in the most carefully-fed cases, when some irritating (undigested) food lies in the stomach, and the pylorus contracts strongly and remains closed. Vomiting follows. The repetition of such a condition gradually induces a more or less continuous spasm of the pylorus, or at least a spasmodic contraction which seriously interferes with the normal emptying of the stomach. It may be that in some cases, as in those recorded by Willoughby Gardner, and by Still, it is possible, solely by careful dieting and the use

* *Brit. Med. Journ.*, vol. ii, 1906, p. 939.

of a food leaving little residue in the stomach, to check this over-action of the circular fibres of the pylorus. Such cases are probably rare, but they support the view that there is no inherent vice in the pylorus. They are rare because a diagnosis could seldom be made at a stage when such treatment would usually be effective. By the time a diagnosis can be made with certainty the spasm of the pylorus is established, and as the result hypertrophy and dilatation of the stomach have followed. We have then to deal with a fastidious, irritable, dog-in-the-manger pylorus, which must be humoured, and soothed, and deferred to in every way. It is not to be supposed that even in marked cases the pylorus never relaxes. At operations it has frequently been found that the hypertrophied pylorus can, and does relax, and will often admit the little finger without difficulty. In many cases the presence of fæcal matter in the stools, and the absence of vomiting for a week or more, show that food is passing through the pylorus. The gravity of the affection lies in the fact that it does not do so in sufficient quantity for the nutrition of the infant. These would appear to be the grounds on which medical treatment may be expected to do good, and, expressed in other words, the aim of the treatment is to restore the function of relaxation of the pylorus which has been in abeyance owing to the more powerful action of the constrictor fibres.

(2) *The food and feeding.*—Although the stomach is much dilated and is capable of holding a large amount of food, small feeds are called for in order to secure complete digestion. Two or three ounces will be found sufficient at a meal, and in bad cases one ounce feeds are advisable. In order to supply a sufficient amount of nourishment the number of meals must be increased. An infant of one month should be fed every two hours, day and night, and in bad cases, when only small quantities are allowed at a time, every hour by day. This may have to be carried on for some months unaltered, but if the progress of the case allows of it, one will naturally try to increase the amount at a meal and feed less often as time goes on. If the infant will sleep for longer intervals at night it should be allowed to do so, but hunger will usually wake it with great regularity. Small quantities, one to four drachms, of plain water or dill water may be given between the feeding times if the child is thirsty or wakeful. The difficulty in the complete digestion of the food in the stomach lies in the proteids and the fats, more especially the

latter. Over and over again I have found the addition of cream or cod-liver oil bring on gastric irritation and vomiting.

If good breast milk is available it is the best food. A chemical analysis of the milk should be made, and one would prefer to have rather a low amount of all the chief solids. The fat should not be more than 3 per cent. The quantity given at a time must be regulated by the duration of the feed—five or ten minutes as the case may be. Unfortunately in many of these cases one finds that suckling has been stopped, in the belief that the milk was disagreeing with the child, and one of the most useful adjuncts in the treatment is lost.

If artificial feeding is employed, cow's milk as usually modified does not act well owing to the heavy curds which remain in the stomach. The foods which have proved most successful in my experience are peptogenised cow's milk, (without added cream,) whey, and Allenburys' Food (No. 1). This last I take as a type of foods which are not in themselves suitable for healthy children, but which are capable of very complete digestion in the stomach. In addition, malt extract, raw meat juice, and grape juice, have been given daily in small quantities. No doubt other foods may be found equally efficacious, and the question of the suitability of the food is to be decided by the state of the stomach contents after washing out.

(3) *Gastric lavage.*—The stomach should be washed out once a day for a prolonged period, and in bad cases this proceeding may be required twice daily for a time. It is quite a simple process in young infants, and if properly carried out leads to no discomfort or disturbance. It should be done when under normal conditions the stomach is empty, *i. e.* two or two and a half hours after a meal, as the case may be. The value of the information gained from the nature of the stomach contents cannot be over-estimated. The wash-out should show a small amount of soft flocculent matter which tends to get less in successful cases as time goes on. If, on the other hand, a larger amount of matter is washed out, or undigested curds, it is clear that the food is not properly digested and will maintain the pyloric spasm. Dr. Cautley has pointed out that in many cases the mere washing out of the stomach systematically will check the vomiting, and that this may prove misleading. This is perfectly true, and therefore one must examine carefully the nature of the stomach contents in order to see whether the residue would be likely

to pass the pylorus. If this residue is of the soft flocculent nature described above one may reasonably expect that it can and will prove non-irritating, and will eventually pass naturally through the pylorus. Other indications of improvement must also be looked for, and will be referred to later.

For the observation of these points, and for the lavage to be of any real value, it is of the first importance that the medical attendant should carry out the proceeding himself, and not entrust it to any nurse, however skilled and experienced she may be. Gastric lavage is not only beneficial to the infant, but is also the means of showing the physician how to regulate successfully the food and feeding.

(4) *Signs of progress.*—(1) The vomiting ceases. This usually follows quickly after lavage and proper feeding have been carried out.

(2) The bowels act naturally. This is a very important sign of progress, for it is in such marked contrast to what occurs in untreated cases. In the latter constipation is the rule, artificial aids *per rectum* being very often required, and the motions contain little fæcal matter. After the medical treatment has been begun the bowels act two or three times a day without artificial stimulation, and the motions are larger and consist of fæcal matter. Coincidently with this, the amount of material present in the stomach two hours after a feed steadily diminishes.

(3) The stomach peristalsis becomes less marked, less strong, and gradually passes off. The complete disappearance of peristalsis may take some weeks or months, but the lessening is usually seen early in successful cases.

(4) The discomfort, pain, apathy, and constant whining of the infant are removed, and are replaced by a placid and comfortable state, in which even smiling is gradually evolved.

(5) The nutrition of the infant is improved. This is manifested in a healthier colour, in increased activity of the limbs, and in a slow gain in weight. It is very important to remember that these infants cannot be fattened rapidly, that any attempt to secure this will probably end in disaster, and that the less weighing there is the better, especially if there are anxious relatives about. The most suitable foods are not fattening ones, and any excess of food is apt to produce disturbance either in the stomach or the bowel. On the other hand the vital powers of the infant can be very much improved, and the disordered functions restored to healthy action by a simple diet, and the fattening materials can be

reserved until a later period. Progress in the infant is usually marked by a slow increase in weight, somewhat fluctuating at first but tending to become steady as time goes on.

(6) *Complications.*—A common complication in the course of treatment is diarrhœa. The bowel in such cases has been out of use for some time, and is consequently unprepared for the food material which the pylorus comes to pass on. Hence probably arises an irritative form of diarrhœa. The best treatment is to reduce the amount of food given by one half, and to keep it so until the diarrhœa has ceased. At the same time half a grain of grey powder may be given twice or thrice daily. When the diarrhœa ceases the amount of food can be again gradually increased. Such diarrhœa is always serious and may prove fatal. When vomiting occurs, no food should be given for two hours in order to rest the stomach. Sips of hot water may be given if the child is thirsty. Intestinal flatulence is to be treated, like diarrhœa, by a temporary reduction in the amount of food.

(7) *Medicinal treatment.*—The use of anti-spasmodic drugs such as opium and the bromides have not proved of the slightest value in my experience. Of course, infinitesimal doses of opium cannot be expected to do any good, but I have pushed the drug to its full physiological effects without any benefit whatever. Nor does it appear likely that the drug would have any special action on the circular fibres of the pylorus and fail to affect the longitudinal fibres to an equal extent. In cases of marasmic infants suffering from this affection, with all the tissues dried up and a constant feeling of thirst, the use of saline injections, both subcutaneously and *per rectum*, has seemed beneficial. From four to ten ounces of normal saline solution can be given daily, and will act as a restorative until the improvement of the pyloric function allows of the entrance of a sufficient amount of fluid into the system by the natural route. In cases in which the infant is much reduced, or has a subnormal temperature, brandy, up to a half drachm daily, will be found of service as a general tonic. The administration of cod-liver oil by inunction, if it be of any value, would appear to be specially suited to the conditions present in this affection. I have only employed it once, and in that case a skin eruption quickly followed and led to its discontinuance.

XXIII.—*Some Cases of Hypertrophic Pyloric Stenosis in Infants.* By Arthur Voelcker, M.D. *Read March* 8, 1907.

THE paper I have the honour of reading before you to-night is based on my experience of seven cases which have been under my care in my ward at the Hospital for Sick Children in the last two years. I propose to give a brief abstract of each case and then to discuss certain of the points which appear to me to bear upon the ætiology and treatment of the condition.

Conclusions drawn from insufficient experience are notoriously fallacious, but I offer my notes as a small contribution to a subject which at present certainly presents many difficulties. I have been enabled by the courtesy of my colleagues and by the kind help of Dr. Reginald Millar, our Registrar at Great Ormond Street, to place before you some results of an analysis of all the cases which have occurred in our hospital since the disease was recognised. Some of these cases have been already recorded.

Abstract of Clinical Notes of Each Case.

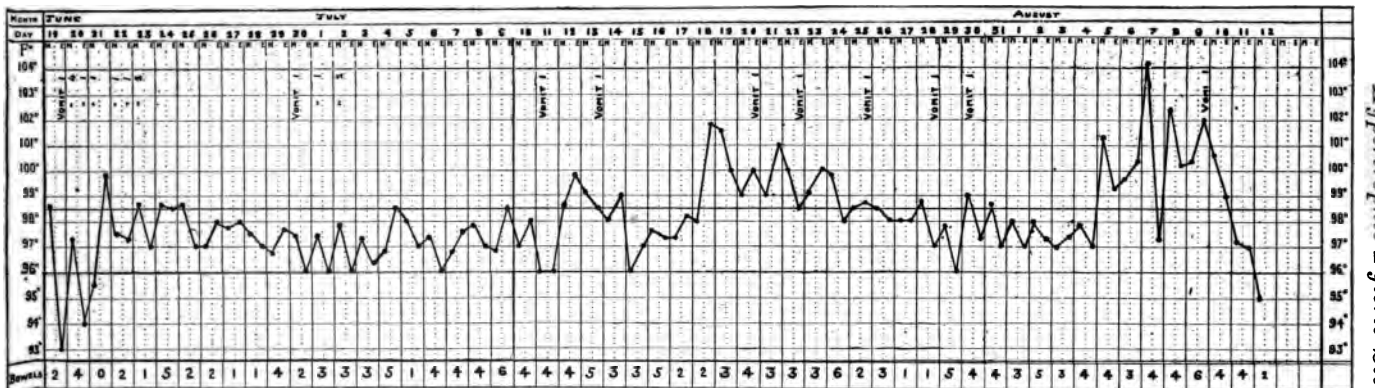
Case 1.—R. T., male, æt. 2 months; was 54 days in hospital: died. No. 6 in the family. Was well for 14 days, when vomiting commenced, at first 5 minutes after feeding, the milk often coming through the nostrils, then the food was retained for nearly 30 minutes before the vomiting began. Breast-fed till 4 days before admission. Bowels very constipated.

June 19, 1905 (see Charts 1 and 2).—On admission: wasted; weight 5½ lb.; temperature sub-normal; fontanelle depressed; no craniotabes. Visible peristalsis, but pylorus not felt. Billowy peristaltic waves seen passing from left to right. Vomits 10 minutes after being fed. Ordered peptonised milk, ½ oz. every half hour; brandy ♏ij every 2 hours.

June 20.—Stomach lavage twice a day. Peptonised milk ℥i every hour.

June 24.—Lavage omitted on 20th, 21st, 22nd, and 23rd; no improvement; resumed on 24th with the effect of diminishing the vomiting. Pylorus now felt. Has lost ½ lb.

CASE 1, CHART 1.



Date. 1905	Diet.	Treatment.
June 19	Peptonised milk, ʒss, ½-hourly, brandy, ♏x, 2-hourly	Gastric lavage every day.
„ 20	Peptonised milk, ʒi hourly	—
„ 23 „ 24	Peptonised milk and barley water, ʒij, āā ½-hourly	Gastric lavage.
„ 24	Peptonised milk and barley water, āā ʒi, every hour	—
July 2	Peptonised milk, ʒi, barley water, ʒss, hourly	—
„ 4	—	Nasal feeding.
July 7	Peptonised milk, ʒij, brandy ♏x, every hour	—
„ 8	Cream, ♏x, in feeds	—
„ 8	Peptonised milk, ʒij, 2-hourly, cream, ♏xv, alternate feeds	℞j. Liq. Strych., ♏ij, 4-hourly.
„ 26	Peptogine milk powders	—
„ 29	Gravy (beef), ʒss, 4 times per day	—
Aug. 9	—	Rectal wash-out and starch enema.

July 1.—No vomiting till June 30. Gained ¼ lb.

July 4.—Only sick once since last note. Bowels open 3 times, motions green. Lost ¼ lb.

July 21.—Has been fed every 2 hours only. No gain of weight. Has only vomited twice in last 19 days. Much curd in gastric wash-out.

July 27.—Has lost 1 lb., but only vomited twice. Motions green and watery.

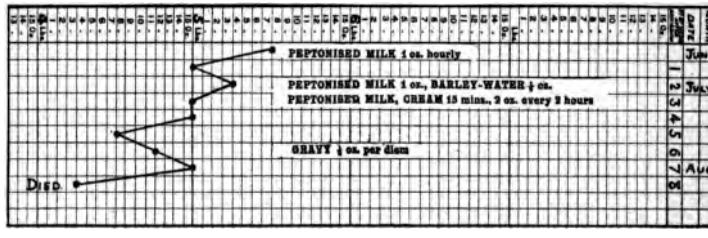
July 28.—Given beef gravy, ½ oz. every 4 hours.

August 1.—Vomited on 30th. Diarrhœa continues, abdomen distended; visible peristalsis; pylorus felt, but in spite of this patient is gaining weight.

August 8.—Diarrhœa worse; temperature 104° F.

August 13.—Death.

CHART 2.



CASE 1.—Weight.

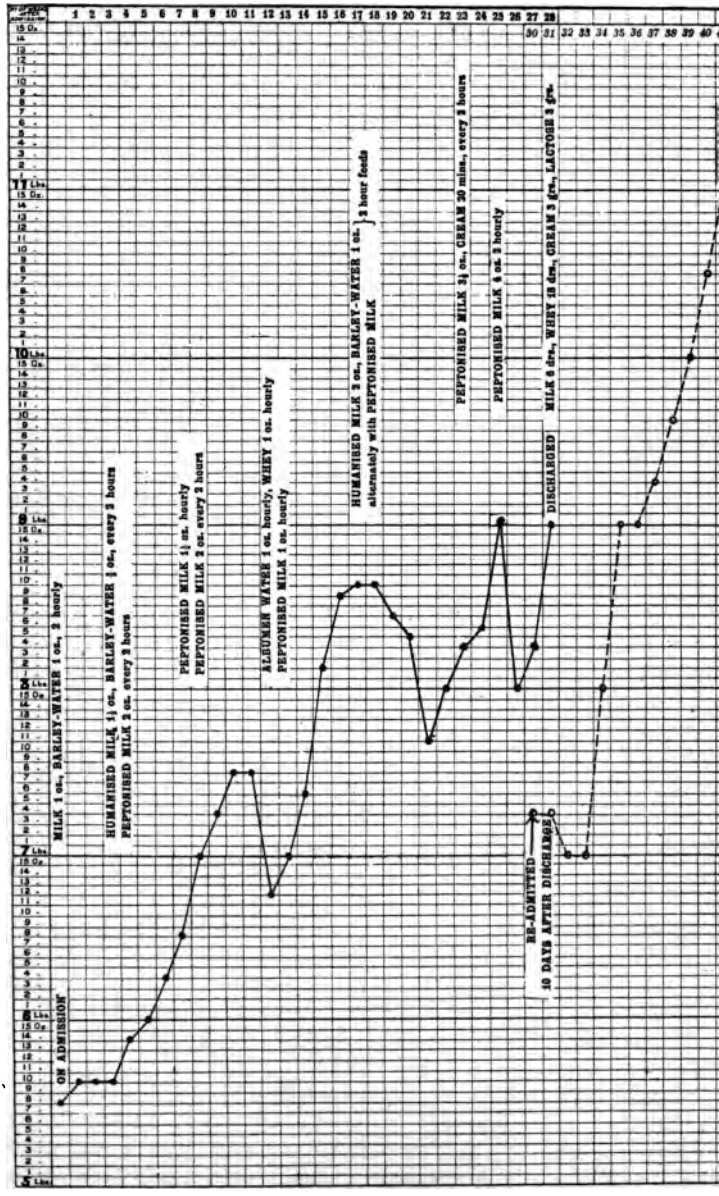
Post mortem.—Pylorus much thickened; hypertrophy of circular fibres of pyloric ring; mucous membrane plicated, but not inflamed; submucosa thickened; pyloric opening allows fluid to pass in either direction; orifice admits a cedar pencil; walls of stomach much thickened; duodenum natural. No abnormalities in rest of body.

CASE 2.—S. L., male, æt. 10 weeks; 189 days in hospital; recovered. Vomiting began when 3 weeks of age. No. 3 in the family; long labour; breast-fed for 7 weeks, then on cow's milk, ½ oz. to water 1 oz. for three days, then on Nestle's milk 1 : 6 of water; markedly constipated.

February 9, 1905.—On admission: poorly nourished; weight 5½ lb; requires circumcision; double inguinal hernia; abdomen distended; visible peristalsis; no pain; pylorus just felt. Vomits soon after a feed; gastric fluid contains HCl, pepsin, peptones, albumoses; egg albumen is not digested by it with any readiness; gastric lavage twice a day; it is

CASE 2.—Weight.

CHART 3.



noted that the wash-out after humanised milk contained more dense curd than after peptonised milk. Under this treatment the vomiting only occurred every other day and the child gained weight.

May 17.—Lavage once daily.

May 22.—Lavage omitted.

May 27.—Abdominal distension relieved by gastric lavage.

During June patient had diarrhœa and vomited about once a day.

In July he regained weight, the peristalsis was no longer visible and the pylorus was not felt.

On August 15 the pylorus was again felt and peristalsis seen.

Discharged August 17, having gained 3½ lb.

Six days later he was re-admitted with severe gastro-enteritis; he had lost 1 lb. 12 oz. He only vomited on two occasions, was treated by gastric and rectal lavage, and fed on humanised milk, yolk of egg, Benger and raw meat juice, and was given citrate of potash.

He rapidly improved, gained weight (weight 11 lb.) and was over-fed after leaving the hospital. When seen at Christmas, 1906, was quite well, but over-fat.

Case 3.—J. C., male, æt. 3 months; 28 days in hospital; died. No. 2 in family. Not well since birth, but only vomited when 1 month of age. Constipated. Breast-fed for three weeks and then on peptonised milk.

February 28, 1905.—Wasted, weight 7 lb. No craniotabes; abdomen tense; pylorus not felt; no visible peristalsis. Vomit contained brown mucus and curd. Blood found in vomit, none in stools. Daily lavage; peptonised milk cream; mist. carminativa. Two days later peristalsis seen and pylorus felt. Not sick. Lost ½ lb. in first week (6 lb. 8 oz.) Ordered humanised milk.

March 13.—Lavage omitted. Vomits once a day.

March 21.—Less vomiting, weight constant (6 lb. 10 oz.). Mist. carminativa omitted.

March 24.—Vomited 3 times yesterday; very collapsed.

March 26.—Only sick once, has gained 1 oz.; weight 6lb. 9 oz.

March 27.—Sudden and fatal collapse.

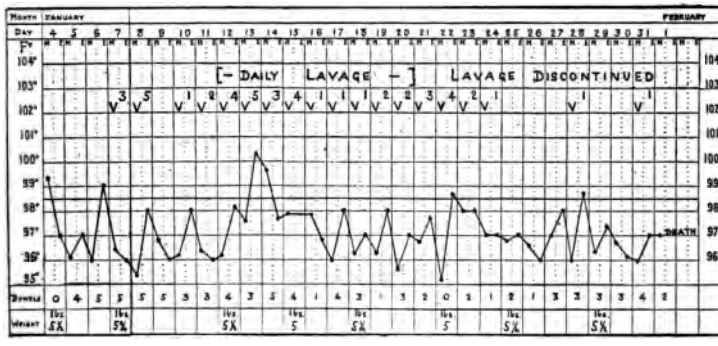
Post-mortem.—Weight 6 lb. Walls of stomach thick, opaque. Pylorus lies far back; admits No. 4 catheter. Pyloric wall (without mucosa) $\frac{7}{32}$ in. thick. Mucous mem-

brane not plicated. Stomach holds 4½ oz., and contains curd and mucus. Some yellow fæces in the intestine. No congenital deformities.

Case 4.—A. F., male, æt. 9 weeks; 28 days in hospital; died. No. 4 in family. Wasting since 1 week of age. Breast-fed 4 days, then on Nestle's milk ʒj, water ʒxij, then on diluted cow's milk. Vomits 5 minutes after being fed. Vomit described by the mother "like as if it had come out of a tap." Motions natural.

January 4, 1906.—On admission: wasted, weight 5lb.

Case 4, Chart 4.



Date. 1906	Diet.	Treatment.
January 5	Cow's milk, ʒiss, barley water, ʒij, every 2 hours	℞ Hyd. c. Cret., gr. ½, t.d.s.
„ 6	Mist. carmin, gr. i ·06	Mist. Ol. Ric., ʒss, t.d.s.
„ 9	Cow's milk, ʒj, boiled water, ʒij, brandy ♏xx, every 2 hours	—
„ 14	Peptonised milk, ʒj, boiled water, ʒij, every two hours	—
„ 17	Peptonised milk, ʒiss, cream, ♏x, water ʒj, every 2 hours	—
„ 22	Alternate feeds of plasmon water	—
„ 23	*Plasmon water, ʒij, cream, ♏x, brandy, ♏xv, every 1½ hours	—
„ 27	Add cream ♏v to each feed	—
„ 30	Peptonised milk and barley water, āā ʒj, cream ♏xv, brandy, ♏xv; alternately with plasmon water, ʒij, cream, ♏xv, brandy, ♏xv ("2-hourly feeds")	—

* Plasmon ʒj, water ʒiv.

4 oz.; no craniotabes; obvious peristalsis; sometimes three tumours seen; pylorus not felt.

January 7.—Nasal feeding (cow's milk 1½ oz. and barley-water 2 oz.) Vomits 3 times a day; bowels open 5 times a day.

January 8.—Vomiting continues; temperature subnormal. By the 12th the vomiting had subsided, but the diarrhœa continued.

January 12.—Gastric lavage; brown-stained (blood) mucus in stomach; has lost ½ lb. Ordered peptonised milk.

January 19.—Gained 4 oz.; less sickness; cream added to milk.

January 20.—Collapsed; omit lavage; more vomiting on 21st, 22nd, and 23rd. Ordered Plasmon water and peptonised milk alternately.

January 26.—Plasmon water, cream and brandy, every 1½ hours.

January 30.—Much less vomiting; gained 4 oz. (weight 5 lb. 8 oz.); 3 stools a day.

February 1.—Died.

Post-mortem.—Weight 5 lb.; stomach folded back; pylorus to left of vertebral column; stomach will hold 3⅛ oz.; no excess of mucus; pylorus 1¾ in. in circumference; pyloric ring (without mucosa) $\frac{3}{16}$ in. thick; mucosa plicated; lumen admits a tube $\frac{3}{16}$ in.; duodenum natural; intestines empty; no congenital deformity. Microscopical examination of the pylorus shows but little change in the mucosa, the submucosa is thickened; the most marked change is in the circular muscular layer, which is much hypertrophied. The longitudinal fibres are hardly at all hypertrophied; some of its fibres interlace with those of the circular coat.

Case 5.—F. R., male, æt. 2 months; 136 days in hospital; recovered. No. 8 in family; difficult labour. Quite well till 14 days of age. Breast-fed 14 days, then with equal parts of cow's milk and barley-water; then on Mellin's food, and on Horlick's malted milk. On admission taking cow's milk and boiled water in equal volumes.

January 25, 1906.—On admission: fairly nourished; weight 9 lb.; no craniotabes; distinct peristalsis from left to right, three distinct prominences seen in the stomach; pylorus felt; constipated.

January 30.—No vomiting for three days.

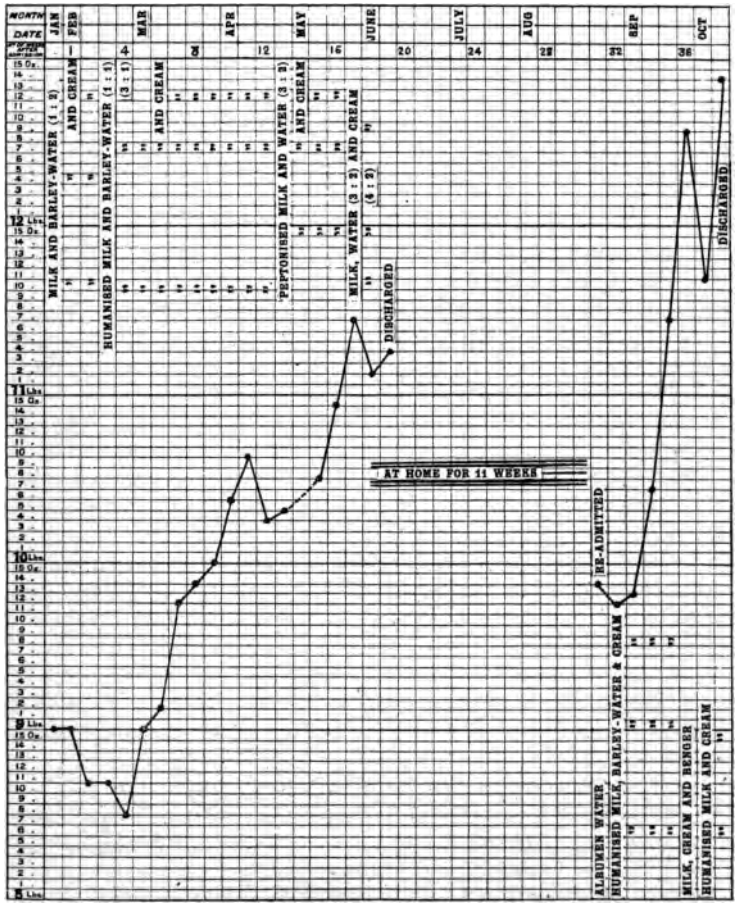
January 31.—Visible hour-glass contraction of the stomach.

February 3.—Given Sanatogen.

February 9.—Daily lavage.

February 14.—Has lost weight; put on humanised milk and barley-water. On this he gained weight.

CASE 5.—Weight. CHART 5.



March 14.—Rise of temperature and vomiting for the last 4 days.

From March 27 to April 24 there was some vomiting, but the weight gradually increased.

On May 12 lavage discontinued, but resumed from May 24 till May 30, on account of vomiting.

The child was sent home, having gained 2¼ lb. in the 20 weeks he was in hospital.

On August 21 he was re-admitted on account of vomiting and diarrhœa, but there was no vomiting or diarrhœa while in hospital. The child gained rapidly in weight, and when discharged on October 8 weighed 12 lb. 14 oz., a gain of 3 lb. in 6 weeks.

Case 6.—D. L., male, æt. 9 weeks; 27 days in hospital; *death*. Vomiting and constipation since 2 weeks of age; Breast-fed at first, then on milk and water; constipated. No. 3 in family.

March 16, 1906.—On admission: thin; weight 7 lb. 8 oz.; no craniotabes; visible peristalsis; pylorus not felt; abdomen distended.

Daily lavage; alkaline mixture 3 times a day. Gradual loss of weight; vomiting frequent; rise of temperature to 101°F. Death on 27th day. Lost 1 lb. 5 oz.

Case 7.—W. C—, male, æt. 14 days; 15 days in hospital; *death*. No. 1 in family; natural labour; weighed 10 lb. at birth. Vomited from the fifth day; breast-fed; constipated. Was sick after 4 or 5 feeds, whether of milk or simple barley-water.

February 16, 1907.—On admission: very wasted; weight 6 lb.; temperature 98·2° F.; abdomen distended; no visible peristalsis; pylorus not felt. Vomits after each feed; is in pain; phimosis is present.

February 20.—Visible peristalsis. An alkaline mixture has not affected the frequency of the vomiting; lavage twice daily. Bromide of potash and Tr. opii ♏½ do not control the sickness.

February 27.—Albumen water ℥ij, Valentine ♏v, substituted for humanised milk without diminishing the vomiting.

March 3.—Diarrhœa for last week; death; weight 5 lb. 9 oz., loss of 7 oz. since admission.

Post mortem.—Slight hypertrophy of the stomach walls; pylorus much thickened, $\frac{3}{16}$ in. thick; dilatation of left renal pelvis.

Remarks.—The existence of hypertrophic stenosis of the pylorus seems first to have been recognised by Dr. Hezekiah

Beardsley, of Connecticut, in 1778, but it is only of recent years that it has been re-discovered in this country by the writings of Dr. John Thompson, 1896, and Dr. Still among others. Never having come across a single case during the three years I was Registrar and Pathologist at Great Ormond Street, I was at one time rather sceptical as to the existence of the condition, but I am now certain that the condition is one which can be recognised both during life and in the *post-mortem* room. I propose to call your attention to certain points illustrated by my cases, and to bring before you the results of an analysis of all the cases (39 in number) which have been in the Hospital for Sick Children in the last ten years; for this information I am indebted to the kindness of Dr. Reginald Millar, our Medical Registrar.

Sex.—All my cases occurred in males, and of the 39 cases in the Children's Hospital 35 occurred in males and only 4 in females. This preponderance of incidence in males must be more than accidental, but it is interesting to note that we have no record of two cases occurring in the same family.

Position in the family.—The existence of hæmorrhages in new-born children, so well demonstrated by Prof. Herbert Spencer in his paper in the *Obstetrical Society's Transactions*, and the possibility of some such hæmorrhage into the pylorus being the cause of the stenosis, made me investigate the above point. In only one of my cases was the child the first child, and of 34 cases at Great Ormond Street 9 were first children.

The nature of the labour.—In 2 of my cases this was described as difficult, but both these cases recovered. Of the 28 cases at Great Ormond Street the labour was normal in 18 cases and difficult in 10.

In no case with difficult labour did symptoms come on within 14 days, and in one case (a breech presentation in which the jaw was fractured in delivery) the symptoms only appeared after six weeks.

These observations, I think, make it impossible that the condition can be properly explained by hæmorrhage occurring at the time of birth, and the microscopical evidence is also quite opposed to this view. The stenosis is partly produced by hypertrophy of the circular muscular fibres and not by scar-formation. Were the hæmorrhage sufficient to produce the stenosis later on by scar-formation, it would cause stenosis by its mass in its recent state, and all the clinical evidence is against this.

In all my cases the children were full-term children, and

of 27 cases at Great Ormond Street I find only 2 were premature children.

Age at which vomiting began.—One case commenced vomiting at 4 days, 1 at 7, 3 at 14, 1 at 21, and 1 at 30 days.

In 37 cases noted at Great Ormond Street 4 commenced at birth (10·8 per cent.); 3 at 1 week; 9 at 2, 11 at 3, and 5 at 4 weeks (70·27 per cent.); 2 at 5, 2 at 6, and 1 at 8 weeks.

These observations tend to disprove the existence of a congenital lesion at the pylorus. If the condition be a congenital stenosis, why do not symptoms develop early in the same way in which we find the vomiting occurring in cases of congenital stenosis of the duodenum? The passage of motions, and, indeed, the frequency of diarrhœa, proves, too, that the stenosis must be spasmodic and incomplete, and in the last place, as I shall point out, the vomiting often ceases for long periods and may or may not recur.

The co-existence of other congenital malformations is rather conspicuous by its absence. One of my cases had a dilated pelvis to one kidney. In 33 cases examined in our *post-mortem* room, a dilated pelvis of one kidney was found in 2 cases (one of Dr. Still's cases and one of mine). Slight webbing of the toes was present in 1 case and a cystic kidney in 1 other case. The co-existence of abnormalities is far too infrequent to justify our attaching any importance to it.

Method of feeding previous to the onset of vomiting.—In 3 of my cases the child was at the breast when the symptoms developed, and I find that out of 37 cases at Great Ormond Street, 21 were at the breast (56·75 per cent.). This would show that the symptoms cannot be ascribed to artificial feeding or merely to the formation of masses of curds which might cause a mechanical obstruction at the pylorus, as the vomiting has occurred when only barley-water has been given.

Survival.—Of my 7 cases, 2 recovered, 5 died; but our hospital records are less cheerful, for out of 39 cases, 5 have recovered, while 34 have died (a mortality of 87 per cent.). In my fatal cases, 1 died at 6 weeks, after being 15 days in hospital; two died at 3 months, after being 4 weeks in hospital; 1 died at 4 months, after being in 4 weeks, and one at 4 months, after being in 8 weeks.

I mention these periods that it may be seen that the fatal cases were under treatment for a sufficiently long time to test the efficacy of treatment.

The fact that these cases survive for so long throws much

doubt on the view that pyloric obstruction is the cause of death. The vomiting is often found to disappear or to diminish markedly towards the end of cases which terminate fatally, while in cases which recover vomiting may be frequent and long continued, and the progress of a case, either to recovery or death, can not be gauged by the vomiting alone.

The vomited material.—Occasionally the vomit is brown from the presence of altered blood, but I have never seen bright blood after either vomiting or lavage. The presence of blood in the vomit is a bad sign. Although curds are not often vomited we may find them in the stomach washings, and they are by no means infrequent in the stools—a point to remember before we accept an entirely mechanical explanation of the causation of symptoms.

The time at which vomiting occurred was generally within five minutes of the ingestion of food. The vomiting was often accompanied by visible peristalsis, the waves moving from left to right in all my cases. Pain sometimes accompanied this peristalsis but not invariably.

Nature of the food.—Vomiting did not appear to be much influenced by the nature of the food. When all milk was stopped and albumen-water substituted, vomiting still continued, and I am inclined to think that the vomiting is the least dangerous of the symptoms of the condition.

I have tried various modifications of milk and have found none so useful as the humanised milk to which barley-water and cream have been added. Peptonised milk gives a smaller curd in the stomach than humanised milk, but the children do not do so well upon it. On the charts of my cases I have noted the various things I have tried, and I have found nothing better than diluted humanised milk. Increase in weight and the character and frequency of the stools, and not the existence or non-existence of vomiting, are our indications of progress.

The condition of the pylorus.—In four of my cases the pylorus was felt at some time during life, and in three, all of which were fatal, it was not felt. It was in some cases felt at one time and not at another, and in one case where it was found markedly hypertrophied I could never feel it during life. From the way in which we find the pylorus tucked away under the liver or behind the rest of the stomach in the *post-mortem* room I think it is not essential that the pylorus should be felt before we can establish the diagnosis of hypertrophic pyloric stenosis.

The gastric contents have been acid in all the cases I have tested with litmus paper. In one case the fluid drawn from the stomach was examined by Dr. Graham Forbes, our Clinical Pathologist, who found HCl, pepsin, peptones, albumoses. The fluid did not digest egg-albumen readily. Investigations are now being made on the gastric contents in cases at Great Ormond Street. The existence of pyloric spasm in adults in cases of excessive secretion of HCl has suggested that the pyloric obstruction may be due to a spasm of the sphincter caused by an excess of HCl in the stomach, but treatment with alkalies (possibly in too small doses) has not been successful. I have been in the habit of administering alkalies, but in one case in which I observed the relation of the vomiting with the administration of an alkaline mixture I did not find that the vomiting was influenced by it.

I am not convinced that an excess of HCl in the gastric secretion will explain the condition, but I think that we shall find in some perversion of the gastric secretion the cause of the pyloric condition. The perverted gastric secretion I believe to be independent of the food supplied, and I believe it causes a reflex spasm of the pylorus, that this is followed by hypertrophy of the circular fibres, but that this mechanical condition is not the only factor is seen by the malassimilation of food which occurs in these cases and which leads to a condition of marasmus. The important part of the disease I believe to be this malassimilation of food and not the prevention of its passage into the bowel. If the pyloric stenosis were the cause of the wasting and of the vomiting we ought to see improvement follow the cessation of vomiting, but this is certainly not always the case. The manner in which these cases so often terminate, either by diarrhœa, by pyrexia, or with an acute attack of vomiting and diarrhœa, or with subnormal temperature and collapse, bears a close resemblance to cases in which marasmus follows on acute gastro-enteritis.

The condition of the bowels.—In 6 out of 7 of my cases constipation was present before admission, but 3 developed severe diarrhœa though 1 of these recovered. I have already pointed out that the occurrence of diarrhœa shows that there cannot be obstruction sufficient to prevent the passage of a considerable amount of food into the bowel.

Weight of child.—This is most important from the point of view of prognosis—the heavier the child the better the prognosis.

In 35 cases at Great Ormond Street the weights were:

Between 4 and 5 lb.	.	2 cases.		
„ 5 „ 6 „	.	8 „	.	1 recovery.
„ 6 „ 7 „	.	14 „	.	1 „
„ 7 „ 8 „	.	6 „	.	2 recoveries.
„ 8 „ 9 „	.	4 „	.	1 recovery.
Over 9 „	.	1 case.		

Of the cases that recovered the weights were: 5½ lb., 6¾ lb., 7¼ lb., 8 lb., 9 lb.

Treatment.—Taking the view that I do, of the nature of the disease, I do not see that surgical interference in the way of stretching the pylorus or establishing another communication between the stomach and bowel, is either necessary or advisable.

Our experience at Great Ormond Street has not been great—5 cases have been operated on but without a recovery.

Lavage of the stomach, by removing secretions which cause reflex spasm of the pylorus, as well as by removing a certain amount of curd, has done good in my cases.

I am in the habit of having the stomach washed out once daily, and then, if the case is progressing, having the lavage on alternate days and so gradually discontinuing it. Apart from the question of food, medical treatment has not appeared to me to influence the progress of the cases. I have been in the habit of ordering an alkaline carminative mixture, with one minim of glycerine of carbolic acid.

Opium has not appeared to me of use except in the treatment of intercurrent diarrhœa, nor have bromides done good. The diet I believe to be the most important item in treatment. Both my cases which recovered were fed on modified cow's milk, which gave better results than peptonised milk.

It must be admitted that the treatment of these cases is very unsatisfactory.

XXIV.—*The Surgical Treatment of Congenital Pyloric Stenosis.* By F. F. Burghard, M.S.(Lond.),F.R.C.S. *Read March* 8, 1907.

WHEN the subject of congenital stenosis of the pylorus was last under discussion in London, namely on December 9, 1902, operation was generally considered essential in nearly all cases, and there was a general consensus of opinion that this should be done quite early. Since then a considerable change has occured. The experience of several observers has shown that cases of undoubted stenosis of the pylorus can be, and have been cured, by medical measures alone. These observations have now become so numerous that the position of surgical interference in this affection is by no means as assured as it appeared to be five years ago. The matter, however, is still full of uncertainty and confusion. Thus at the last debate on the subject held in the section of Pediatrics at the Toronto Meeting of the British Medical Association in August, 1906, one speaker stated that the condition was almost always fatal unless treated by operation, and that the aid of the surgeon should be invoked almost invariably; while another expressed profound disagreement with this view, stating that he had seen 8 well-marked cases during the last ten years, all of which had got well under medical treatment alone.

It cannot be denied that the position reached in the debate just referred to is unsatisfactory, and it is to be hoped that the discussion this evening will help to reconcile these diametrically opposed opinions, and to clear up the most important question now remaining in connection with this affection, namely, what cases, if any, of congenital stenosis of the pylorus should be operated upon, and at what period of the disease is operation required. Personally I have no doubt that many of these cases can be cured without recourse to surgery, and I cannot help thinking that the number will increase as knowledge of the disease extends and the practitioner recognises it and employs appropriate treatment as soon as it arises. Treatment by operation must always remain dangerous however much its details may be improved, owing to the tender age of the patient operated upon. Therefore there must be a tendency to resort to medical treatment

to the exclusion of surgery, and therefore the operation results are likely to be less good in the future than they have been in the past. It is not unreasonable to suppose that the cases coming under the care of the surgeon in the immediate future will be chiefly those that are too bad when they first come under notice to allow medical treatment to have any chance or those that have gone downhill in spite of medical treatment.

I propose to confine the few remarks I shall make to-night to a consideration of the 16 cases that I have operated upon. These have all been subjected to dilatation of the pylorus, the various steps of which I propose to describe. It is perhaps as well to do this, as similar communications have been made by Mr. Clinton Dent upon the subject of pyloroplasty for this condition, and by Mr. Harold J. Stiles upon gastro-jejunostomy.

The only contribution I can make to the discussion of the question whether medical or surgical treatment is the more suitable for these cases is the statement that, taking all my cases together, the direct operative mortality is 5 in 16, or 31·25 per cent. The causes of death were shock in 2 cases, perforation of the duodenum in 1, some enteritis in another, and no ascertainable cause in the remaining case. This is the direct operation mortality, but if it be considered necessary to include in the mortality of cases treated surgically those that die within three months of the operation, another case must be added, viz. that of a child that died nearly three months after operation in convulsions. This gives a total death-rate of 6 in 16 cases, or 37·5 per cent.

It is to be hoped that physicians here to-night who have had these cases under their care will tell us not only how they have treated them, and how many have recovered by medical measures alone, but also how many have died while under medical treatment, so that we may be able to form an estimate of the mortality of the condition when the surgeon has no part in it. In connection with the morbid anatomy of the affection it may be well to call attention to the fact that there was never any real occlusion of the pylorus in any of my cases. A No. 8–10 catheter could be passed through every one of them with ease. The statement often met with in reports of cases, that the pylorus would barely admit a probe, can only, therefore, be accounted for by *post-mortem* rigidity. It was also always noticed that the consistency of the tumour varied considerably, the pyloric ring alternately contracting and relaxing.

Preliminary treatment.—Whenever possible special prelimary treatment is employed for at least two days before the operation. The child is fed by nutrient enemata, and a saline infusion of 4–6 oz. is given daily beneath the skin of the abdomen, thorax or back. The thighs and axillæ are left available for a similar purpose after the operation.

Immediately before the operation the extremities are wrapped in cotton-wool, and gamgee tissue envelopes the thorax. The child is laid upon an india-rubber hot-water bottle, and a mackintosh and a warm folded blanket are interposed between, otherwise the child may be scalded, as the covering of the bottle becomes saturated with lotion during the operation. The two chief essentials for success are a high temperature in the operating room, which should never be less than 70° F., and rapidity in operating. The whole thing from start to finish should not occupy more than twenty-five minutes, and, in order to economise time, everything should be laid out ready to hand, and the washing out of the stomach, which is an essential point, should be done just before the anæsthetic is administered, and not after. The children do not object to it in the least, and will suck contentedly at the tube all through the procedure, which may sometimes be a very tedious one. I also purify the skin, and arrange the towels around the seat of operation before the anæsthetic is commenced. The surgeon and the assistant then stand ready to commence directly the anæsthesia is sufficiently advanced.

The purification of the skin is effected by thorough washing with ether soap and hot water, after which a sterilised pad, wet with a solution of 1 in 500 biniodide and spirit, is laid over the actual site of incision. A sterilised towel is laid over the abdomen, and a slit 3 in. long is cut in its centre. The anæsthetic is then begun, and as soon as the child is sufficiently under, an incision is made in the middle line downwards from the ensiform cartilage for nearly 2 inches. This incision is ample, and only exposes the liver down to its free margin; this is important, as by observing it one may prevent protrusion of the intestines. The skin is always extremely thin, and care is required in the first incision, as it is easy to wound the liver with the first stroke of a sharp knife.

After the peritoneal cavity has been opened, the finger is hooked round the free margin of the liver, which is pulled up, and thus allows the distended and hypertrophied stomach

to present in the wound. It is caught with forceps, and along with the pylorus is pulled well out of the wound, and a small cloth is packed around it so as to keep back the rest of the intestines and allow the operation to be done outside the abdomen. A horizontal incision about half an inch in length is now made through the stomach wall with stout scissors; this should be situated quite 1 inch from the pylorus and mid-way between the two curvatures. The edges of this incision are seized in two pairs of catch forceps, and held up by the assistant. The gas at once escapes from the stomach, which collapses and thereby facilitates the rest of the operation. No fluid is likely to escape from the stomach if this has been washed out thoroughly before the operation is commenced. Any small amount of fluid remaining may be prevented from escaping by pulling the lips of the incision well upwards.

The enlarged pylorus is now grasped with the fingers of the left hand, and a four-bladed screw dilator is passed through it from the incision in the stomach. Owing to the attachments of the duodenum, the dilator must be kept quite vertical throughout the operation, or else there is risk of perforating the anterior wall of the duodenum, an accident that happened to me on one occasion. The dilator is screwed up very slowly, until either a pre-determined amount of dilatation is arrived at (namely, enough to admit the little finger), or until the peritoneum on the anterior surface of the pylorus shows a tendency to crack. At least ten minutes should be occupied in doing this. When dilatation appears to be sufficient the dilator is withdrawn, and the little finger is insinuated through the pylorus. After this a No. 10 red rubber catheter is passed down the duodenum, and 6 oz. of peptonised milk with a drachm of brandy at the body temperature are administered through it, the pylorus meanwhile being compressed around the catheter to prevent regurgitation. This pressure is maintained as the catheter is withdrawn, and the wound in the stomach is then rapidly closed, first with a continuous suture of stout catgut, taking up all the coats of the organ, and subsequently with fine continuous Lembert suture, also of catgut. The parts are finally douched with hot saline solution, and the stomach and omentum dropped back into the abdomen, and the liver allowed to fall into place. This at once acts as a shutter, keeping everything in place, and allows the abdomen to be closed in two layers, the deep suture of catgut taking up the peritoneum

and the linea alba, whilst the superficial one of fine silk takes the skin only. The child is then given $\frac{1}{2}$ minim of liq. strych. hypodermically, and a subcutaneous saline infusion of 6 oz. into the axilla.

For the first twelve hours the child is fed *per rectum*, and one or two saline infusions are given. Strychnine may also be employed should it be required. After this, drachm doses of peptonised milk, each containing a few drops of brandy, are given by the mouth every hour, and the dose is steadily increased as the child improves.

Difficulties and dangers.—The prevention of the first great danger, namely shock, has already been dealt with. If the operation be done as recommended above there is little to fear from shock, except in very emaciated children. I have lost two cases from this cause, but in both it was quite open to question whether it was justifiable to operate at all, so desperate was their condition. Although shock was marked in about half of the remaining cases, it could hardly be called desperate, and in the rest there was only a moderate amount. One of the most remarkable points about this operation is the ease with which these tiny babies stand it.

The chief danger of the operation is undoubtedly splitting the pylorus. This I have done twice, but it is not nearly so serious an accident as might be thought. In the first case, which was also the first of my sixteen cases, it was due to the incision of the stomach being made too close to the enlarged pylorus. As the dilatation proceeded the stomach incision split right through the anterior surface of the pylorus. It was, of course, recognised at once and sewn up, and the child made an uninterrupted recovery. I shall have to refer to this case in another connection later. In the second case the rupture occurred through half the length of the pylorus on its posterior aspect, and was not recognised at the moment it occurred. The child was fed into the duodenum in the usual way, and it was only when the abdomen was being sewn up that milk was noticed extravasating into the lesser sac of the peritoneum. The gastro-hepatic omentum was at once torn through, the rent in the pylorus sutured, and the milk washed out of the lesser sac. Unfortunately this child died on the sixth day quite suddenly, after having made uninterrupted progress without vomiting or bad shock, and *post mortem* no cause could be found for death. The incisions had healed perfectly (indeed, there was no visible trace of them), and there was no sign of peritonitis.

The only other accident that has happened during the operation is perforation of the duodenum by one of the blades of the dilator. This, I regret to say, was due to the fact that the operation had to be hurried, as the child had a piece of the catheter, used for lavage, lodged in its stomach, and this had to be found and removed before proceeding with the dilatation. No doubt the dilator was not held quite vertical, and, unfortunately, the lesion was not noticed and sutured at the time, otherwise the child would have probably recovered, as it did very well until it was fed by the mouth for the first time, when it suddenly collapsed and died.

The last, and in some respects the greatest, difficulty connected with the operation is prolapse of the intestines or omentum. Those who have experience of abdominal operations in children know how this delays closure of the wound, and in these young and delicate children it may be enough to turn the scale against them. I find, however, that by keeping the incision high enough, and using the liver as a shield, the difficulty is got over.

The only remaining point that I wish to raise is the question of which operation is the most suitable for these cases. Extended experience leads me to think that the choice should lie between the operation I have just described and pyloroplasty. It is true that gastro-jejunostomy has given fairly good results in the skilful hands of Mr. H. J. Stiles and others, but I cannot help thinking that until it can be shown that the two first-mentioned operations are definitely more fatal and less effectual than gastro-jejunostomy, the latter should not be considered. Apart from the fact that we are still in the dark as to the physiological effect of gastro-jejunostomy upon the alimentary economy, it must, I think, be admitted that in ordinary hands it will be a slower, more difficult, and more dangerous operation than either dilatation or pyloroplasty. Of course, this applies to the posterior operation. The anterior form is quite inadmissible; irrespective of the greater risk of a vicious circle, it leaves an anatomical condition of affairs that no one would wish a child to carry about with it for the rest of its life.

With regard to the choice between dilatation and pyloroplasty, perhaps one may say in these cases, as in so many others, that the best operation is that which the operator has become most proficient in. My reason for rejecting pyloroplasty was furnished me in my first case, to which I have already referred, in which the pylorus was split right through

whilst dilatation was proceeding. It was a matter of the most extreme difficulty to suture the split pylorus at all; the most I could do was to insert a single imperfect layer of sutures, which I could hardly think would hold water at all. I felt sure that the child would die, but as a matter of fact it made a perfect recovery. One of my colleagues who performed pyloroplasty in a similar case a short time after had a similar experience. The thickness of the pylorus in all cases upon which I have had to operate has made me very doubtful of my ability to perform a successful pyloroplasty, and, as I can hardly hope to obtain better results from it than from the method I use, I have so far never attempted it. Mr. Clinton Dent, however, who has used this method largely, speaks highly of it, and has had excellent results.

Lastly, recurrence of symptoms has been urged as a reason for rejecting dilatation. *A priori* one might expect this, and we know that such cases have happened. In none of the cases upon which I have operated has there been any recurrence.

Full tables of the cases referred to in this paper are appended:

Sixteen Cases of Congenital Stenosis of the Pylorus Treated by Dilatation.

Case 1.—Edward J., æt. 7 weeks, admitted to King's College Hospital September 22, 1902, under Dr. Still. Began vomiting at 3 days of age, while breast-fed. Marked peristalsis; pylorus felt; weight at birth 9 lb., on admission 6 lb. 4 oz. Operation September 28, 1902. Pylorus very large; longitudinal incision into stomach; pylorus dilated up to No. 10 Hegar. This led to a splitting of the pylorus, as the stomach incision was made too close to this structure. The greatest difficulty in sewing up the split pylorus, owing to its thickness. Operation lasted twenty-five minutes. Child did perfectly well, and on January 25, 1904, weighed 2 st.

Case 2.—Stanley P., æt. 5 weeks. Admitted to King's College Hospital December 4, 1902, under Dr. Still. Vomiting from birth; operation December 7; weight 6 lb. 8 oz. Operation similar to above, except that the incision in the stomach was further away from the pylorus, and so the latter was not split. Dilated up to No. 17 Hegar. Child did perfectly well, in spite of varicella, and went out March

10, 1903, weighing 8 lb. 9 oz. On June 22, 1903, weighed 18 lb.

CASE 3.—Harold C., æt. 8 weeks. Admitted to King's College Hospital under Dr. Still, March 11, 1903. Vomiting from second day. Weight at 3 weeks 10 lb. 8 oz., at 8 weeks 6 lb. 13 oz.; marked peristalsis; pylorus felt. Operation March 12. Stomach much dilated; vertical incision into stomach wall; pylorus dilated up with a special four-bladed dilator till index finger passed easily through. Fed into duodenum before suturing stomach incision. Did well and went home in June weighing 11 lb. 2 oz.

CASE 4.—John S., æt. 10 weeks. Admitted to King's College Hospital under Dr. Still, May 15, 1903. Vomiting from 4 weeks; visible peristalsis; pylorus felt; weight 6 lb. 12 oz. Operation May 25, 1903. As in preceding case with special dilator; dilated until peritoneum began to crack. Child very collapsed at first, but rallied well, and went home June 19 weighing 7 lb. 12 oz. Weight on February 6, 1904, was 17 lb.

CASE 5.—John J., æt. 8 months. Admitted to the Children's Hospital, Paddington Green, under Dr. Guthrie. Operation July 20, 1903; weight 11 lb; pylorus overstretched as above, and child fed into duodenum. Recovered without a bad symptom, and went out on August 11, weighing 11 lb. 11 oz.

CASE 6.—Leonard H., æt. 3 months. Admitted to King's College Hospital under Dr. Still, June 24, 1903. Vomiting since birth; peristalsis; pylorus felt; weight 6 lb. Operation September 30, 1903, as in last case; some collapse, but recovered well and went out January 6, 1904, weighing 9 lb. 14 oz. On June 18, 1904, weighed 19 lb.

CASE 7.—Master H., æt. 7 weeks. Seen with Dr. Still on October 20, 1904. Vomiting since birth; visible peristalsis; pylorus felt. Operation October 20, 1904, as in last case; child rallied well from the operation and gained weight, but eventually died in a convulsion early in January, 1905.

CASE 8.—Master B., æt. 7 weeks. Seen with Dr. Still. Vomiting from fourth week; peristalsis marked and pylorus

felt. Operation September 17, 1904, as in last case; pylorus very thick indeed; recovery quite uneventful; is a very healthy child now.

Case 9.—Elizabeth A., æt. 8 weeks. Admitted to the Children's Hospital, Paddington Green, under Dr. A. Morrison, September 28, 1904. Vomiting since birth; peristalsis visible and pylorus felt. The child was miserably ill and emaciated, and much doubt was felt as to the propriety of operating, but the loss of weight was progressive and it was decided to try. Operation October 12, 1904, as above; weight 5 lb. 14 oz. The operation was done with great rapidity, and a large feed was given by the duodenum, and the abdominal cavity filled up with saline solution to neutralise shock. In spite of this the child never really rallied from the operation, and died next morning. The *post-mortem* showed the pylorus intact; there was no damage to the mucous membrane beyond a very slight ecchymosis. Death was due to pure shock in an exceedingly feeble subject.

Case 10.—Edward M., æt. 7 weeks. Admitted to King's College Hospital December 30, 1904, under Dr. Still. Marked peristalsis; pylorus felt; weight 6 lb. 4 oz. Operation January 5, 1905; weight 6 lb. Operation as above, done very rapidly on account of the child's serious condition; the entire thing lasted 25 minutes. Child rallied well from the shock, but died suddenly 27 hours after operation—on being fed for the first time by mouth. *Post mortem*, there was a clean rent in the first part of the duodenum. In this case the operation was hurried on and its performance hurried through on account of the detachment of the end of the catheter used for gastric lavage; by some mischance the end got broken or bitten off, and remained in the stomach. This had to be extracted, and, as part of the operation was occupied in doing this, the dilatation was done a little more rapidly than usual. It was not a split of the pylorus, but the end of the dilator had gone through the duodenum.

Case 11.—Master M., æt. 5 weeks. Operation July 14, 1906; seen with Dr. Still. Weight at birth 6 lb. 1 oz., at time of operation 4 lb. 11 oz.; very bad case, vomiting from birth; had not been having any appropriate treatment; usual operation; for 12 hours after the operation slept comfortably, with pulse of 90: three hours later got restless and pulse quickened.

In spite of saline infusion and rectal injection he got worse, had convulsions, vomited dark-coloured fluid, and died 18 hours after operation. He had had several convulsions before operation.

Case 12.—Male, æt. 6 months. Vomiting began a fortnight after birth and persisted in spite of breast feeding; weight on admission 8 lb. Admitted to Paddington Green Children's Hospital April 20, 1906, and treated by dieting and washing out stomach; usual operation, July 28, 1906, with dilator. Half the length of the pylorus was ruptured at its duodenal end and on its posterior aspect. The child was fed into the duodenum, and when sewing up the abdomen, milk was noticed in the lesser peritoneal sac; this was torn through, the rent in the duodenal end of the pylorus closed, and the milk washed out; abdomen closed in the usual way. A good deal of shock for first few hours, but the child rallied well after saline infusion. Fed *per rectum* for 12 hours, then by mouth; practically no vomiting; did perfectly well until August 3 (sixth day), when it refused food, and died suddenly. *Post mortem.*—All wounds soundly healed; no peritonitis; no apparent cause of death (Dr. Emery).

Case 13.—Master H., æt. 5 weeks. Seen with Dr. Archibald Garrod and Dr. Kendal, of Chiddingfold, September 13, 1906. Vomiting began at three weeks, when the child was at once weaned. Peristalsis had only been noticed a few days before the child was seen. Weight 6 lb. 2 oz.; has lost 14 oz. in the last fortnight. Stomach washed out twice daily for the last week, and the vomiting much improved, but loss of weight was progressive; marked peristalsis, and the pylorus is distinctly palpable. Seeing the child was in good condition it was decided to try careful dieting and washing out. On October 28 the child had wasted very considerably, and had lost 1 lb. since he was last seen. He looked pinched and miserable, and it was therefore decided to operate at once. Dilatation was performed, the pylorus being stretched with the screw dilator until it would admit the little finger. The operation lasted twenty-five minutes. The child went back to bed in very good condition, and recovered well from the shock. The next day he had had a good night with practically no vomiting, and was taking rectal feeds well. The following day, October 29, there was considerable intestinal irritation, the bowels acting four times, the motions

being loose but not offensive; the child appeared to be very well. At 8 o'clock, however, there was an attack of violent intestinal colic, accompanied by two loose motions. From this he rallied, but had another attack a few hours later in which he died. *Post mortem.*—The union in the wound and the stomach was perfect; there was no peritonitis, and the condition of affairs in the abdomen was perfectly satisfactory. No cause of death was found.

Case 14.—Master F., æt. 6 weeks. Seen with Dr. Still, December 2, 1906. Vomiting began on thirteenth day and had lasted ever since. For the last ten days he has had his stomach washed out. Weight 5 lb. 2 oz.; he has lost 15 oz. in the last week; visible peristalsis and much enlarged pylorus. Immediate operation was decided upon and done in the ordinary manner, except that Hegar's dilators were used instead of the screw instrument. Operation lasted twenty-five minutes; there was practically no shock after it. The child was fed by mouth at the end of twenty-four hours; he took food well, gained 10 oz. in the first week; from that time he has made steady progress.

Case 15.—John J., æt. 7 weeks. December 8, 1906. Vomiting from 4 days of age; very persistent. Weight 5 lb; has lost 18 oz. in the last week; has been treated for the last ten days by washing out; visible peristalsis; pylorus easily felt; looks very pinched and emaciated. Early operation was decided on in view of the desperate condition of the child. Operation was done on December 11, the intervening period having been occupied in administering rectal feeding and saline infusions; pylorus very large and hard; screw dilator employed until pylorus would admit the little finger; operation lasted thirty minutes. Child considerably collapsed after operation, but recovered well in the course of the next eight hours; vomited once, bringing up a small quantity of blood, but not afterwards; was fed by the mouth in fifteen hours, took food extremely well; gained 17 oz. in the next first fortnight, and is now well.

Case 16.—Master Y., æt. 14 weeks. Seen December 18; was a fine, healthy boy at birth, and did well for the first three weeks; vomiting since then, first at intervals, but for the last three weeks almost incessantly; has had no treatment beyond drugs; exact loss of weight not known, but apparently

marked; child looks desperately pinched and ill; very great dilatation of stomach; pylorus large and hard. Operation was performed next day; lasted twenty minutes; screw dilator employed; child exceedingly collapsed after operation and throughout the night, but he rallied after three administrations of strychnine and two saline infusions; next morning appeared bright and well; was given food towards mid-day, which was retained. This child did well except for a slight attack of diarrhœa on the third and fourth days; has now gained 3 lb. since the operation, and is apparently well.

XXV.—*A Modification in the Method of Applying Prepared Filigrees of Silver Wire to the Cure of Large Inguinal Herniæ in Males.* By LAWRIE MCGAVIN, F.R.C.S.Eng. *Read April* 12, 1907.

ALTHOUGH I have used the term "modification" in deference to Dr. Willard Bartlett, whose filigree I look upon as one of the greatest advances in the recent surgery of abdominal hernia, I have been unable to find any record of the application of the filigree to an uncomplicated case of inguinal hernia in a male patient other than my own.

Bartlett himself has published a case in a woman,* and quite recently Wiener has reported one in a male,† but in this case the difficulty which suggested the necessity of a modification to me, viz. the presence of the spermatic cord, was absent, the patient having undergone removal of the cord and testis at a previous operation. This case, therefore, resolved itself into one much resembling the operation as done for ventral hernia, or inguinal hernia in a woman.

The first case in which I employed a prepared filigree for the cure of inguinal hernia, was that of a seaman, æt. 63. I have fully reported it elsewhere,‡ and will therefore only say that the filigree I used was a single one, that its introduction was an easy matter on account of the long loose cord, which I was able to draw outwards round the outer end of the filigree, and that it was oblong in shape, measuring three inches by one and a half. Although the case so far has done well and there is no sign of bulging, I am a little doubtful of the ultimate integrity of the inguinal tissues external to the filigree, as this region is to my mind hardly sufficiently guarded. This fact, and the experience gained in my second case, suggested to me the necessity of some modification in the manner of introducing and shaping these filigrees.

This second case, also reported, was one of extensive recurrence in a young seaman after a so-called "radical cure." I found that the filigree I had made could not be introduced as in the last case owing to the tension of the cord, for, being three inches long, it exceeded the distance from the internal abdominal ring to the pubic spine. I was therefore compelled to divide it and place one half internal to the ring,

* *Annals of Surgery*, 1903.
† *Ibid*, 1906.
‡ *Practitioner*, 1906.

and the other external to it, making the outer half overlap the inner by about half an inch, the cord lying between them. I also experienced some difficulty in fitting the inner half into its place in the angle formed by Poupart's ligament with the margin of the rectus muscle at its insertion, and was obliged to double over the loops of the filigree at the pubic end.

Since this case was done I have adopted the method in the more perfect manner detailed below for the following reasons. The presence of the spermatic cord renders the implantation of a filigree in such a manner as to defend the entire inguinal region a matter of some difficulty. Either the cord must be drawn outwards round the outer end of the filigree so as to permit the latter to lie in front of, and so defend, the internal abdominal ring, or the filigree must lie in front of the ring and the whole length of the cord in the canal. The former method will only be found possible where, as in my first case, the patient is old and wasted, with a long lax cord: and even then, owing to the limited amount of elasticity of the latter, an undefended space is still left external to the filigree. The second method provides a position for the filigree which is very uneven, and in which it is loose and easily shifted, and leaves an undefended space at the point of emergence of the cord at the pubic end. Besides being difficult to carry out satisfactorily, it abolishes the valvular mechanism of the inguinal canal.

In order, therefore, to afford complete defence to that part of the inguinal region which lies external to the internal abdominal ring, and at the same time to preserve the valvular arrangement of the inguinal canal, I have resorted to the following method, which I have found simple and effective.

The filigrees which I now employ are made for me in two sections, an iliac and a pubic, and apart from their shape are produced in a manner similar to those invented by Bartlett for abdominal herniæ, and which I demonstrated to this Society in 1903. The pubic section is made one and a half inch long, three-quarters of an inch wide at the inner, and one and a half at the outer end; it is thus trapezoid in shape. The iliac section may be of any suitable length, usually two to two and a half inches; its outer half is square or oblong, while its inner half corresponds in shape to the outer half of the pubic section. The method of introduction is as follows:

The fullest exposure of the parts having been obtained by the division of the aponeurosis of the external oblique muscle

from the external to a point well beyond the internal abdominal ring, the sac is freed and disposed of in the usual manner. The cord, after being completely freed from any adhesions to the pillars of the external ring and the conjoined tendon, is held aside, and the peritoneum is gently peeled back from the deep surface of the conjoined tendon and the back of Poupart's ligament. As soon as any oozing has been controlled the gap thus made is extended as much as possible by the use of retractors, and the pubic section of the filigree is laid on the peritoneum, the points of the loops being neatly disposed beneath the edges of the tendon and Poupart's ligament. These latter structures are then approximated by Bassini's method, the filigree being thus completely buried.

The cord is now replaced, and a director being passed beneath them, the fibres of the internal oblique and transversalis muscles are cleanly divided from the internal ring outwards for a distance of about two inches. These divided edges are turned upwards and downwards, and again the peritoneum is peeled back from their deep surface. A bed is thus formed for the iliac section of the filigree, the outer square or oblong portion of which is laid upon the peritoneum, while the inner trapezoid portion covers the first inch of the cord as it emerges from the internal ring. The cord, it will be seen, now lies, for the first inch of its course, between the two sections of the filigree. Finally, the cut margins of the external oblique aponeurosis are united over the whole in the usual manner, and the skin incision is closed.

It is unnecessary to fix these sections in any way as they will be found to be retained in position by the mere apposition of the muscles. It will be seen that this method permits of the most perfect defence of the inguinal region, the size of the iliac section being determined solely by the requirements of the particular case. For the sake of uniformity I now use the same gauge of wire for all filigree operations, viz. No. 28 Standard Wire Gauge, and the same number of loops to the inch, viz. eight. In the case of females there is, of course, no object in the use of filigrees made in two sections, as the difficulty of the presence of the cord does not arise; a filigree, therefore, of the same shape as the pubic section just described, and measuring three or four inches in length, three-quarters of an inch wide at the pubic, and two to two and a half at the iliac end, will be found to satisfy the requirements of most cases.

Two points of especial importance in the use of these

filigrees are the necessity of a perfectly dry wound, and the avoidance of drainage. Most of the cases requiring filigree-implantation are complicated by extensive matting of the parts from recurrence, or from truss-pressure, and therefore oozing of blood and serum must be expected; drainage in this situation is, however, likely to result in infection. Should such an accident occur, convalescence will be unduly prolonged; but, as I have elsewhere pointed out, removal of the filigree will probably not be necessary, nor is there reason to suppose that recurrence will result, as would undoubtedly be the case if no filigree had been employed.

Although I should strongly advocate this method as being the only one to which the term "radical cure" can honestly be applied, I do not practise it as a routine method, reserving it rather for cases which are obviously incurable by the ordinary operation, as shown by the large size of the hernia, the fact of recurrence, the presence of feeble muscular development, or the advanced age of the patient.

My cases done in the last two years are not numerous, being six in number, but all were genuinely incurable by ordinary means. So far the results are perfectly satisfactory, although I am aware that more time is still required for proof of complete cure. Judging, however, from the results I have obtained during the past three and a half years in the treatment of so-called incurable abdominal herniæ by the use of Bartlett's filigree, I have no hesitation in considering them to be examples of "radical cure" in the truest sense of the term.

In only one case has there been any interruption in convalescence: this was the first of the series already referred to, in which a single filigree was used, and in which drainage was employed. Owing to an unfortunate omission the drain was not removed sufficiently early, and some slight infection took place. The final result was, however, very satisfactory.

No complications referable to irritation of the cord by the filigree, such as testicular or inguinal pain, epididymitis, orchitis, or atrophy of the testis have been noted.

The most recent case in which I had occasion to employ this method was that of a seaman, æt. 43, a native of Trinidad, whose hernia was a very large one, resulting from a kick in the right groin. It was complicated by a large vaginal hydrocele which was possibly the chief factor in the descent of the hernia, and measured 12 in. in length, its greatest circumference being 18½ in.

At the operation the sac was found to contain a large mass of small intestine with its mesentery, and the hernial gap measured nearly three inches by two; the absence of omentum bore out the man's statement that the hernia was of recent occurrence, and the result of an injury. The cord and internal abdominal ring were displaced outwards. It had been my intention, if possible, to save the cord and testis, and implant the filigree as above described, in two sections; this, however, I found to be impossible, owing to the size and attachments of the hydrocele. These structures had unfortunately to be sacrificed, but I used the two sections of the filigree, placing the pubic section on the peritoneum, and the iliac section, after approximation of the conjoined tendon and Poupart's ligament, on the internal oblique muscle opposite the internal abdominal ring. The aponeurosis of the external oblique was then sutured over the whole length of the canal.

In spite of the persistently dirty habits of the patient, his dressings being constantly soaked with urine, the result has been good, and he returns to Trinidad with the inguinal region firm and sound.

XXVI.—*Two Cases of Lateral Ventral Hernia treated by the Implantation of very large Filigrees of Silver Wire.* By Lawrie McGavin, F.R.C.S.Eng. *Exhibited April* 26, 1907.

CASE 1.—A woman, æt. 38, was admitted to the Hospital for Women in December, 1905, for the removal of a very large and adherent broad ligament cyst. Three weeks after operation she came under my care, having developed a large tense swelling in the right iliac region, with a temperature of 103° F. This, on being opened, discharged a quantity of very thin pus, and later nothing came from the wound but pure urine. It was now clear that the right ureter had been tied in. A few days later an attempt to find the point of ligature failed, owing to the density of the adhesions, in dealing with which a fæcal fistula resulted. The kidney was removed through the loin. The patient now became extremely ill, and a gangrenous patch, the size of a five-shilling piece, appeared around the mouth of the sinus. Eventually she recovered and was discharged, but a year later returned with a hernia at the site of operation, the gap measuring 5 in. by 3 in. The whole of the old cicatrix being removed by an elliptical incision 11 in. in length, it was found impossible to separate the peritoneum from the deep surface of the external oblique aponeurosis; the inner portion of this aponeurosis was therefore separated from the outer portion by a curved incision; external to this incision it was possible to carry out the separation. The divorced strip of aponeurosis was now sutured to the cut edge of the posterior sheath of the rectus muscle, and upon this, as a bed, a silver wire filigree, measuring 8 in. by 4 in., was laid, the inner loops being disposed deep to the rectus muscle, and the outer deep to the oblique muscles. The remains of the rectus muscle were now brought out of the sheath and sutured, as far as possible, to the oblique muscles, over the filigree. This caused a weak spot at the middle line of the abdomen, and necessitated the opening of the sheath of the left rectus muscle, which was drawn across the middle line to meet its fellow, where it was sutured. The skin and superficial fascia were closed over all by thirty-five Michel's clips. Primary union followed, and no belt has been worn since convalescence.

Case 2.—This patient, kindly sent me by Sir Alfred Fripp, was admitted to Guy's Hospital in the early part of 1906 for fulminating appendicitis and general peritonitis. She first came under the care of Mr. Arbuthnot Lane, who opened the abdomen in the iliac region and above the pubes. These wounds were drained for four days, and were then lightly sutured. A few days later fæces appeared on the dressings, and a complete fistula became established. This appears to have been in the upper portion of the bowel, and the patient rapidly wasted. From this time she came under the care of Sir Alfred Fripp, who made four separate attempts to close the fistula by suture, and finally, when she was almost moribund, succeeded in closing it by resection of six inches of intestine. She recovered and was discharged. Three months later she was re-admitted, under Mr. Lane, for a very large hernia of the appendical cicatrix. This was closed and she went out, but in the course of another three months she was again the subject of a very large hernia, for the cure of which she was sent to me. On measurement the gap measured 7 in. in length by 3 to 4 in. in width. The bowels were adherent to the skin, and she was completely incapable of any exertion, as on standing up the larger part of the abdominal contents prolapsed into the sac, causing nausea and vomiting. In January of this year I excised the whole scar by an incision 13 in. in length, and separated a large number of adhesions. So much of the peritoneum had to be sacrificed that it was found impossible to close the abdomen. After every effort a gap remained in the peritoneum and fasciæ which measured 3 in. by 1½ in. Into this gap was drawn a loop of small intestine and a portion of the cæcum; these were sutured together and sewn into the gap. A bed was thus made for the filigree, consisting of the posterior sheath of the rectus muscle internally, small intestine and cæcum in the centre, and peritoneum externally. Upon this bed the filigree of silver wire, measuring 9 in. by 4 in., was laid. An attempt was made to bring the atrophic rectus muscle into apposition with the oblique muscles over it, but was not very successful. Finally, the skin and superficial fasciæ were united over the whole by forty-two Michel's clips and four salmon-gut tension sutures. Primary union resulted. The patient has worn no belt since her convalescence.

XXVII.—*Acute Solitary Suppuration in the Psoas and Iliacus Muscles.* By W. G. Spencer, M.S. *Read April* 12, 1907.

THERE has been a good deal written upon suppuration in the psoas and iliacus muscles, some of it under the term *psoitis*, as well as under the subject of *iliac swellings*.

Acute suppuration has frequently occurred secondary to puerperal parametritis, by direct extension. The same remark applies to suppurative appendicitis and peri-nephritis; it has likewise followed acute osteomyelitis of the spinal column or ilium, as distinguished from chronic tuberculous abscess. There are also cases of puncture and bullet-wounds which have set up suppuration in the muscle; moreover, suppuration has followed ligature of the iliac artery.

I pass over these subjects and come to acute solitary suppuration—its relation to infection and to strain or actual rupture.

The case which called my attention to these questions is as follows:

S. H. was a healthy schoolboy, æt. 13, who made no complaint of an accident, nor could his mother learn that he had met with one. There had been no tuberculous disease in the family. On returning from school at mid-day on February 7 he limped, complaining that it hurt him to move his leg, and that he felt pain over the right ilium, between the crest and the great trochanter. He went to bed that afternoon, but it is not known how he slept. On the second day of the illness he did not complain of much pain, but gradually relapsed into delirium. Dr. Hook, of Clapham, was not called to the patient until three days from the first complaint, when he recognised the presence of a right iliac swelling and sent the patient at once to the hospital, where I saw him soon afterwards.

The boy was then in a low muttering delirium, with a pale face, a brown, furred, dry tongue, the temperature 105° F., the pulse 116, the respiration 30. The chest was normal, also the abdomen except for the swelling; all his bones and joints were sound, also his ears.

He lay with the right thigh flexed nearly to the abdomen, neither everted nor inverted, but it could be straightened out

without exciting the patient; there was no sign of hip-joint or of spinal disease.

The right psoas and iliacus muscles formed a tense swelling which became more prominent when the thigh was extended, and which did not relax under the anæsthetic. A bimanual examination discovered no abdominal nor pelvic disease, only the swelling as above. There was no external swelling of the ilium in the region where the boy had first felt the pain.

I made an ilio-lumbar incision, and pushed the cæcum forwards and inwards off the swelling, to which it had no pathological adhesion. On incising the sheath of the psoas septic fluid oozed out, but the muscle substance was not much altered, nor was there any distinct fluctuation. On thrusting in blunt-ended forceps I reached an acutely suppurating focus deep in the iliacus, in the venter ilii, which had come into contact with the bone. The bone, however, was not involved; there was no sign of extravasated blood from rupture. From 30 to 60 c.cm. of pus escaped, not more; a tube was then inserted. An examination showed that the pus contained a pure growth of *Staphylococcus pyogenes aureus*. He was given two 10 c.cm. injections of anti-staphylococcic serum (brand, Burroughs, Wellcome & Co.), but neither the operation nor the serum had any influence. Some râles appeared in the chest, and the patient died within four days of making the first complaint.

A most thorough *post-mortem* examination was made the next day by Dr. Hebb; the vertebræ and ilium were cut through, the intestinal canal, including the appendix, the spinal cord, hip-joint, pleura, pericardium, heart, ears were all particularly examined, and found normal. In the lungs was some early septic broncho-pneumonia, but there were no abscesses. Only the right psoas and iliacus muscles were affected by acute septic myositis, but the actual excavation was not more than 5 cm. in diameter, and had merely come in contact with the bone without invading it. In the suppurating muscle, in the blood, and in the lungs, the only organism cultivated was *Staphylococcus pyogenes aureus*.

Turning now to the recorded cases and selecting those running an acute fatal course, followed by a *post-mortem* examination, and putting aside those secondary to extension above referred to, then we reach a category where a slight strain has been followed by fatal suppuration, in some of which also a distant septic focus has been discovered.

WITTMAN, "Akute Suppurative Psoitis," *Jahrb. für Kinderheilk.*, 1875, viii, 369.

A boy, æt. 8, had had a chronic ear discharge, but it had stopped for a week or two, when he fell at play and walked home lame. He was thereupon seized with acute suppuration in the right psoas muscle, which was followed by right-sided septic pneumonia, and he died, without any bones or joints becoming involved.

Other cases might be quoted which likewise may be taken as metastatic from a distant old-standing focus.

But there are other cases in which a definite strain or severe rupture has been followed by acute fatal suppuration, and yet there has been found *post mortem* no distant septic focus to explain the infection.

WETTENHEIMER, "Psoitis," *Jahrb. für Kinderheilk.*, 1890, xxx, 365.

A healthy boy, æt. 13, had a slight fall on the hip followed by acute septic inflammation, and he died in eleven and a half days. *Post mortem* there was found suppuration in the psoas and iliacus muscles wholly destroying the psoas and beginning to extend to the sartorius, but nothing else.

CLARKE, S. I., "Psoitis and Peripsoitis; their Pathology and Differential Diagnosis," *New York Med. Journ.*, 1885, ii, 582.

A young man slightly strained himself roller skating, and afterwards had a temperature of 105° F. The fever was first called rheumatic, then a boggy swelling appeared above the pubes, from which foul pus was let out. At the *post-mortem* there was no intestinal or other lesion, but the suppuration had been confined to the psoas muscle. The vertebræ had been bared, but were otherwise healthy.

BOKAI, see WITTMAN, "Akute Suppurative Psoitis," *Jahrb. für Kinderheilk.*, 1875, viii, 369.

A boy, æt. 10, fell off a three-foot wall upon his face and knees and limped home. After four days he could not walk at all. Acute suppuration appeared in the left psoas muscle, followed by a pustular eruption, bilateral septic pneumonia and death. At the *post-mortem* the left psoas and iliacus muscles were found changed into thin, dirty, brown pus and sloughs, which continued under Poupart's ligament to the insertion of the muscles, and the pus was there tending to

track downwards into the thigh. Beyond this there were recent septic abscesses in the lungs but nothing else.

MARCANO, "De la Psoite Traumatique," 1878, Paris (Tract in the Med-Chir. Library), Obs. xviii, p. 62.

An Artilleryman, æt. 20, in the gymnasium alighted flat on his feet from the height of half a metre, which caused him a sharp jolt, and he forthwith felt a severe pain in the umbilical region. But it was only after four days that he was compelled to go into Hospital. He was there found to be suffering from a severe fever and a distended and painful abdomen, which was freely leeched. The inflammation spread down the left cord to the scrotum, and after the eighth day pus began to escape by way of the leech bites. The man died on the thirteenth day. Suppuration was found to have commenced in the left psoas and iliacus muscles, and thence to have spread to the abdominal wall and scrotum outside the peritoneum, which was quite free. There had been no intestinal lesion and nothing more was noted.

There are recorded many less severe cases which have got well after timely incision.

As for the treatment of the severest type, of which mine is an instance, I am afraid we must await the production of a more powerful anti-staphylococcal serum.

Considering the number of cases in which a slight strain has been mentioned one may be inclined to think that the boy under my care may have also strained himself, but at any rate it gave rise to no complaint, and there was no interval in which he walked lame before the acute fever set in. The question of a strain has been much discussed in the puerperal cases, whether the strain occured during the labour, or during the puerperium when turning in bed, or upon getting up.

As to the origin of the infection, it is held by many at the present time that *Staphylococcus albus* is present in healthy tissues, and need not interfere with the healing of a wound. Such a view would seem to run counter to the conjecture that a mild saprophytic staphylococcus may in a suitable nidus suddenly develop into a most virulent parasite.

Another explanation gains support from cases of acute suppuration in other muscles and of acute septic osteo-myelitis. Although no distant septic focus is revealed by a complete *post-mortem* examination, yet a septic infection may have

occurred days or weeks before through a scratch, finger-prick, patch of impetigo, etc. Although the superficial lesion may have quite healed, the organisms have entered the blood-stream, but are held in check so long as they continue within the circulation. The organisms remain potentially virulent, so that, when, as the result of a strain, extravasation of some blood occurs, the organisms immediately develop. Hence symptoms of acute septic inflammation, such as a rigor, may appear within a few hours.

XXVIII.—*A Note on the Value of Anti-Colon Bacillus Serum.* By G. H. Makins, C.B., F.R.C.S., and Percy Sargent, F.R.C.S. *Read April* 12, 1907.

THE following notes have been put together as a preliminary contribution to what we trust will be an inquiry into the value of a specially prepared multivalent antitoxin in cases of infection with the colon bacillus.

The serum was prepared at the Wellcome Research Laboratories, at the instance of Dr. Dudgeon, in consequence of his work with Mr. Sargent* on the bacteriology of peritonitis. That investigation was held by the observers to prove conclusively that in the great majority of cases of peritonitis of intestinal origin it is the colon bacillus which is the chief cause of the acute or fatal symptoms. It was shown that the main factor in the resistance of the peritoneum to this infection is that of intra-peritoneal phagocytosis, and it was suggested in what manner this natural safeguard is brought into play in the cases which recover. There remains, however, the problem of how to assist or supplement that process in cases where the natural powers of resistance are defective, and an attempt to supply this want is offered by means of an antitoxin prepared from several different strains of the colon bacillus obtained from cases of acute peritonitis and other sources.

Several different brands of serum have been prepared. The following brief account of the preparation of one brand has been kindly furnished by Dr. H. H. Dale, of the Wellcome Research Laboratories:

A horse was immunised by receiving;

Fourteen injections of 10 c.c. of 7 days' broth culture of 8 strains of *B. coli* from cases of puerperal fever, at intervals of 7 days;

Seven injections of 5–20 c.c. of a washed culture of a *B. coli* of unknown origin, at intervals of 10 days;

One injection of 5 c.c. of 3 weeks' growth in lactose broth of 7 strains of unknown origin, 7 strains separated by Dr. Dudgeon from cases of peritonitis, 8 strains from cases of puerperal fever.

* Dudgeon and Sargent, *The Bacteriology of Peritonitis*, 1906.

In all cases the cultures injected were killed by the addition of 0·1 per cent. chinosol.

Dosage.—This must necessarily be empirical, but the probability is that not less than 20 c.c. should be given, and repeated at intervals of from 8 to 12 hours. We have seen no ill-effects follow the administration of as much as 40 c.c. at a time.

Recoveries, 9 = 36 per cent.

Diffuse spreading peritonitis from more or less localised abscess, cases 1, 16.

Diffuse peritonitis with no localisation, cases 2, 10, 11, 12, 14, 17, 24.

Case 1.—The slowing of the pulse was the main feature.

Case 16.—*B. coli* had been found alone in the exudate at a distance from the abscess, a condition which has been shown by Dudgeon and Sargent to be almost uniformly fatal. But the good effect here must be discounted by the fact that there had been some attempt at localisation before the onset of the diffuse peritonitis—a condition which has also been shown by Dudgeon and Sargent to be more favourable than when the diffuse peritonitis starts from an absolutely free appendix.

Case 2 was one in which the result of the serum was marked by an almost immediate feeling of improvement, cessation of vomiting, and slowing of the pulse-rate.

Case 10 was one in which the serum was given on the third day after operation because no improvement was taking place. It was followed by immediate relief of symptoms.

Case 11.—Relief of general toxæmic symptoms as well as a fall in both temperature and pulse-rate after each injection.

Case 12.—Relief of general toxæmic symptoms.

Case 14.—Distinct improvement in a child desperately ill, extremely restless, and with "black vomit." Child less restless after each injection.

Case 17.—Effect marked both on pulse and temperature, as well as on general symptoms. In this case Gram-negative bacilli had been found in the peritoneal exudate.

Case 24.—Effect decidedly good. Vomiting ceased, which had been "black"; infection became localised.

The fatal cases, 16 = 64 per cent.

Case 3.—Slowing of pulse, relief of toxæmic symptoms; localisation effected; survived twelve days.

Case 4.—Marked improvement at first, cessation of vomiting; localisation assisted; survived five days.

Case 5.—No effect, but serum not given until after a second

Case No.	Sex.	Age.	Disease.	How long ill.	Operation.	When serum begun.
1, N. D.	F.	8	Appendicitis with diffuse peritonitis spreading from partly localised abscess	8 days	Appendicectomy, sponging, drainage	Day of operation
2, C. F.	M.	17	Appendicitis with diffuse peritonitis, recrudescence of peritonitis 5th day	14 hours	(1) Removal of perforated appendix, sponging, drainage; (2) reopened and sponged again	Day of second operation
3, R. M.	M.	11	Gangrenous appendicitis with diffuse peritonitis	48 hours	(1) Removal, sponging, drainage; (2) 10th day, drainage of abscess on left side	Day of first operation
4, K. P.	F.	12	Perforated appendix, pelvic abscess, and diffuse peritonitis	3 days	Removal, sponging, drainage, lavage of stomach	Day of operation
5, H. S.	M.	20	Appendicitis with diffuse peritonitis spreading from local abscess	12 days	(1) Drainage; (2) reopened next day, appendicectomy, sponging, drainage	After second operation
6, E. S.	F.	10	Imperfectly localised abscess with diffuse peritonitis	14 days	Drainage	8 hours after operation
7, J. S.	M.	9	Gangrenous appendicitis, diffuse peritonitis, imperfect localisation of an abscess	4 days	Removal, sponging, drainage	Day of operation
8, T. W.	M.	6	Appendicitis with diffuse peritonitis	6 days	Removal, lavage, drainage	Day of operation
9, E. W.	M.	21	Gangrenous appendicitis, leaking abscess, diffuse peritonitis	20 hours	Removal, dry sponging, drainage	Day of operation
10, F. M.	M.	29	Perforated appendix, diffuse peritonitis	36 hours	Removal, dry sponging, drainage	3rd day, as no improvement was taking place
11, P. C.	M.	21	Gangrenous appendicitis and diffuse peritonitis	Within 24 hours	Removal, dry sponging, drainage	Day after operation
12, H. R.	M.	26	Gangrenous appendicitis, diffuse peritonitis	4 days	Removal, dry sponging, drainage, subsequent enterolysis (4 days later) for obstruction by adhesions	9th day

Dosage.	Apparent effect.	Results, etc.	Surgeon.
60 c.c. in 40 hours, 2 injections	Steady slowing of pulse-rate. None on temperature. Relief of toxæmic symptoms	Cured, opsonic index not raised by the injections	Mr. Makins
40 c.c. in 48 hours, 2 injections	Steady slowing of pulse, cessation of vomiting, marked feeling of improvement	Cured	Mr. Sargent
80 c.c. in 3 days, 3 injections	Slowing of pulse, relief of symptoms, appeared to effect localisation	Died after second operation, opsonic index low throughout as compared to healthy men	Mr. Ballance
45 c.c. in 3 days, 3 injections	Marked improvement at first, but vomiting recurred on 4th day, and death occurred 5th	Died. *P.M.*—Pus localised, general peritoneal cavity healthy, cloudy swelling of viscera	Mr. Makins
45 c.c. on 2nd day	None	Died 3rd day	Mr. Makins
20 c.c. single injection	None, child moribund when serum given	Died in 48 hours	Mr. Pitts (Mr. Nitch)
280 c.c. in 13 days, 9 injections, maximum single dose 60 c.c.	Appeared to assist localisation, temperature not above 100·8° for 7 days, pulse remained rapid throughout	Died 20th day after evacuation of perisplenic abscess, both films and cultures of peritoneal exudate showed *Bacillus coli*	Mr. Clutton (Mr. Nitch)
50 c.c. within a few hours, 2 injections	None, patient moribund when serum given	Died within 12 hours, films of exudate showed bacilli and cocci, cultures — *Staphylococcus albus* only	Mr. Robinson.
60 c.c in 48 hours, 3 injections	None, but death delayed until 10th day	Died	Mr. Makins (Mr. Nitch).
190 c.c. in 5 days, dosage 20—30 c.c.	Marked improvement at once	Cured	Mr. Ballance (Mr. Nitch).
40 c.c. in 2 days, 2 injections	In a few hours patient much easier, pulse and temperature fell to normal, second dose given as pulse and temperature were rising, again both fell to normal	Cured	Mr. Makins.
60 c.c. in 3 doses	Much more comfortable, complicated by basal pneumonia	Cured	Mr. Makins.

Case No.	Sex.	Age.	Disease.	How long ill.	Operation.	When serum begun.
13, R. H.	M.	17	Gangrene and perforation of appendix, local abscess with spreading-ing peritonitis	7 days	Removal, dry spong-ing, drainage	Day of operation
14, R. H.	M.	3	Perforated appendix, diffuse peritonitis	3 days	Removal, dry spong-ing, drainage	Day of operation
15, M. G.	F.	17	Appendix abscess, spreading peritonitis	6 days	Drainage	24 hours after operation
16, W. A.	M.	27	Appendix abscess, subcæcal and pelvic, also diffuse peritonitis	9 days	Incision and drainage	Directly after operation
17, P. D.	M.	8	Appendicitis, diffuse peritonitis, gangrene, no perforation	6 days	Removal, dry spong-ing, drainage	Day after operation
18, T. W.	M.	6	Appendicitis, diffuse peritonitis	6 days	Removal, lavage, drain-age	Immediately after operation
19, L. C.	M.	6	Appendicitis, diffuse peritonitis	4 days	Cœliotomy, drainage	Immediately after operation
20, F. P.	M.	36	Appendix abscess, later subphrenic abscess	1 week	Drainage	5th day after operation
21, T. M.	M.	12	Gangrenous appendicitis, diffuse peritonitis	4 days	Removal, dry spong-ing, drainage	Day of operation
22, W. T.	M.	53	Appendicitis, diffuse peritonitis	3 days	Removal, dry spong-ing, drainage	Immediately after operation
23, F. T.	M.	11	Gangrenous appendicitis, diffuse peritonitis	2 days	Removal, dry spong-ing, drainage	2 days after operation
24, K. H.	F.	16	Appendicitis, perforation, diffuse peritonitis, exacerbation during an apparently subsiding attack of appendicitis of 11 days' duration	Within 24 hours	Removal, dry sponging	10 hours after operation
25, A. W.	F.	30	Appendicitis, diffuse peritonitis, later subphrenic abscess	2nd day	Removal, dry spong-ing, drainage	Soon after operation

Dosage.	Apparent effect.	Results, etc.	Surgeon.
30 c.c. in 3 doses within 24 hours	Relief, comfort, and sleep secured without drugs	Died 21st day, loculated peritonitis	Mr. Makins.
70 c.c. in 4 doses	Always less restless after injections, child extremely ill (black vomit), but steady improvement	Cured	Mr. Makins.
60 c.c. in 2 doses within 12 hours	Very marked improvement in the toxic symptoms after each dose, pulse slowed and vomiting ceased	Died, loculated peritonitis	Mr. Sargent.
25 c.c., 1 dose	Improvement, but evidence bacteriological rather than clinical, exudate at a distance contained *Bacillus coli*, pus of abscess *B. coli* and a *staphylococcus*	Cured, rheumatism 14 days later	Mr. Makins (Mr. Nitch).
60 c.c. in 5 doses, initial dose 20 c.c., rest 10 c.c. twice daily	General improvement, on one occasion temperature fell from 100·4 to normal, and pulse from 124 to 104 after an injection	Cured, intra-cellular Gram-negative bacilli in exudate	Mr. Clutton (Mr. Nitch).
30 c.c. in 2 doses	None, child moribund when serum given	Died within a few hours	Mr. Robinson.
50 c.c. in 2 doses at 9 hour intervals	None, child moribund	Died within a few hours	Mr. Battle (Mr. Adams).
95 c.c in 4 doses, 12 hour intervals	No effect on pulse or temperature, possibly arrested localisation of a spreading peritonitis	Died	Mr. Robinson.
1 dose of 20 c.c.	None, desperate case	Died	Mr. Ballance (Mr. Nitch).
150 c.c. in 3 doses at 12 hour intervals	Certainly relieved the toxic symptoms, of which delirium was a prominent one	Died 3rd day	Mr. Sargent.
Single dose of 20 c.c.	None	Died	Mr. Sargent (Mr. Nitch).
65 c.c. in 24 hours, 3 doses	Marked improvement, vomiting ceased (had been black), abscess opened 8 days later	Cured	Mr. Wallace.
20 c.c. doses	No effect on pulse or temperature, but infection localised down, and she survived a fortnight	Died	Mr. Corner.

operation, the first limited to drainage only under the impression that the abscess was local.

Case 6.—Single small injection when child moribund.

Case 7.—Appeared to assist localisation in a case where the colon bacillus was the only organism found both in films and cultures of the exudate. Survived four weeks..

Case 8.—No effect; child moribund; 6 days' diffuse peritonitis when serum given.

Case 9.—No effect except, perhaps, assisting a localisation; death delayed until tenth day.

Case 13.—Relief of toxæmic symptoms; sleep secured without drugs; assisted localisation; death delayed until twenty-first day.

Case 15.—Very marked improvement after each dose, vomiting ceased, and pulse slowed; localisation apparently effected. Died of loculated peritonitis.

Case 18.—No effect upon a child desperately ill with six days' diffuse peritonitis and moribund when serum given.

Case 19.—No effect, moribund child; similar to Case 18.

Case 20.—No effect except possibly assisting localisation.

Case 21.—No effect, but this was a single small injection in a case of widespread peritonitis of four days' history.

Case 22.—Distinct relief of toxic symptoms immediately after injections.

Case 23.—No effect, but only a small single dose given as a last resort two days after operation.

Case 25.—No effect except assisting in localisation. Death delayed for a fortnight.

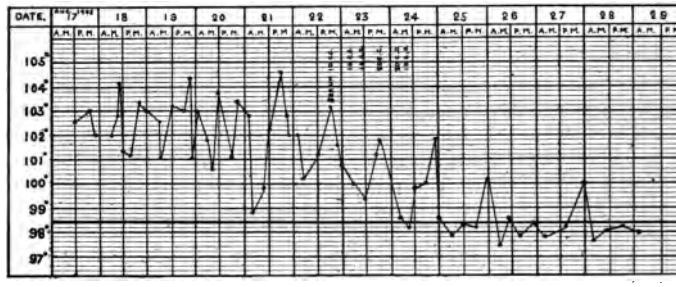
Thus six cases—6, 8, 18, 19, 21, 23—should be subtracted from the fatal ones as unfair test cases; insufficient dosage combined with probable inability on the part of the patient to absorb and make use of the antitoxic bodies.

All the rest in which the body was able to absorb the serum showed some improvement; toxic symptoms were relieved; localisation of the infection was in many cases effected, and death was delayed in many instances beyond the time usual in such cases.

The first trial of an anti-coli serum at St. Thomas's Hospital was made upon a case of acute infection of the urinary tracts by the colon bacillus. The case, which was under the care of Dr. H. G. Turney, presented some remarkable features, and the apparent effect of the serum was very striking. The patient, a healthy young man, æt. 18, gave the following history: Twelve days before admission he was com-

peting in some athletic sports, when, in attempting a high jump, he strained his right loin, but not badly enough to prevent him from taking part in a subsequent race. Three days later he began to have severe continuous aching pain in the back, and rapidly became very ill. Frequency of micturition was a marked symptom; he would, with pain, pass a small quantity of urine every hour, and at times this was accompanied by some bright red blood. When admitted he was tender in the loins, especially on the right side, and the abdomen was rigid. The tongue was dry and brown, and the temperature 103° F.; on the day after his admission he had a rigor and vomited. The urine was acid, contained pus and some blood-cells. Bacteriologically tested it gave a pure

CHART 1.



culture of a colon bacillus. There was a leucocytosis amounting to 19,000. The blood-serum gave a definite, though slight, agglutination reaction with the colon bacillus in a dilution of 1 in 20.

For four days the temperature ranged irregularly between 104·4° and 101° F., and on the fifth day 10 c.c. of the anti-coli serum were injected subcutaneously. Next day he received 40 c.c. in three doses, and on the third day 30 c.c. in two doses. From the time that the serum treatment was begun the temperature steadily fell, reaching the normal on the seventh day. The effect on the general and local conditions was no less definite. The pain, rigidity and tenderness subsided; the tongue became clean, the frequency of micturition disappeared, and the patient became speedily better. There still continued to be a large quantity of pus in the urine, and ten days later the temperature again rose and the symptoms returned. Three

more doses of serum were given, with the same result as before, and the patient made a steady recovery. The specific agglutinative reaction of the blood-serum remained the same after as before the injection.

Conclusions.

No such brilliant results can be claimed for the serum as are obtained in the case of diphtheria, but in a few isolated examples, which really form a large proportion considering how little the serum has as yet been employed, it must be admitted that a directly curative influence has been observed. In several other instances, also, the effect has been such as to justify its more extensive trial.

It must be emphasised, moreover, that the serum has been employed only in cases of the worst type, in which the nature of the infection and the condition of the patient excited the gravest apprehension.

The most striking features observed, after its use, are the general improvement in the patient's comfort, the sequence of quietude or sleep, a definite fall in the pulse-rate and the temperature, either temporary or continued, and in many cases the relief from vomiting. It is noteworthy that in two of the successful cases "black vomit," usually a fatal prognostic sign, had continued for more than twenty-four hours.

A local influence in an increased tendency to early formation of adhesions in the abdomen has also been observed, to which we are disposed to ascribe the prolongation of the course of the disease even in the fatal cases.

The above results appear to us to have proved that the method of treatment exerts a definite influence on the disease, and that it is in no respect harmful or dangerous.

These conclusions are supported by the opinion of an independent observer whose views will carry great weight, namely, Dr. A. K. Gordon,* of Manchester. Dr. Gordon writes as follows:

"I have given serum No. E140 in three cases of puerperal sepsis, all of which appeared to be due to *Bacillus coli* as shown by the presence of that organism in cultures taken from the interior of the uterus, care being taken to eliminate vaginal organisms. In each case *Bacillus coli* appeared in repeated cultures taken on successive days. In all the illness was of a very severe type; one patient died, and in this case

* *Personal communication.*

I performed abdominal hysterectomy more or less as a last resort. From the uterus in that case cultures taken at different layers in its substance all showed *Bacillus coli.*

"Of the two cases that recovered I am certainly of the opinion that the serum was distinctly useful, in fact, the impression on my mind was that recovery was due to the serum, as I had not seen anything like it in similar cases where no serum at all or anti-streptococcus serum only (given empirically) had been used. It is true that in each case the uterus was curetted and packed, but I have had now quite a large number of cases which have been treated in this way without any serum at all. In one of these two cases, however, the group of symptoms which one usually attributes to serum, viz. joint pains, pyrexia, and an urticarial rash, were so very prolonged and severe that I am not sure that the remedy was not worse than the disease.

"I should add that as far as my experience goes the serum made from puerperal cases only seems to yield as good results and to give rise to fewer serum phenomena."

In the cases at St. Thomas's Hospital the symptoms of joint pains, rashes, etc., were only observed to occur in one case (16).

The serum has also been tried in a few chronic cases of colon-bacilluria, but without effect. In fact, it seems to be of no use unless there are symptoms of acute toxæmia present.

XXIX. — *Gastro-Enterostomy for Chronic Gastric Ulcer; healing of Ulcer verified at Post-mortem Examination twenty-eight months later.* By James Sherren, F.R.C.S. *Read May* 10, 1907.

IT is the experience of most surgeons that gastro-enterostomy entirely relieves the symptoms of chronic gastric ulcer in the majority of cases. But it rarely happens that an opportunity arises of minutely examining the condition of the stomach months or years after the operation. In the case which I am about to describe I was able to examine the condition of the stomach twenty-eight months after I had performed gastro-enterostomy for chronic gastric ulcer.

The case seems worthy of publication for I have been unable to find record of a similar examination. Paterson, in his *Hunterian Lectures*, states "that although as yet we have no pathological evidence that gastro-jejunostomy brings about healing of a gastric ulcer, we have clear clinical evidence that this is the case." Mayo Robson and Moynihan mention that they "have had the opportunity of examining the anastomosis months after the original operation, and in one case over three years later." The opening had remained satisfactory in size. But no mention is made of the condition for which the operation was performed.

Thomas C., æt. 46, was admitted to the Poplar Hospital under the care of my colleague, Dr. Cecil Wall, in October, 1904.

He was a spare, sallow man, and had been suffering from gastric trouble for six years. His illness began with pain after food, relieved by vomiting; shortly after the onset of symptoms he was treated in an infirmary for neurasthenia (according to the account given by the patient) for nine months. At the end of this time he was well enough to resume work as a porter, but the symptoms recurred after a short time. His condition varied from time to time, improving with rest and diet, relapsing on resuming work; for more than nine months previous to admission to Hospital he had been away from his work and had spent most of the time in bed, unable to take solid food and suffering severe pain after the ingestion of the lightest diet.

On abdominal examination the stomach did not appear to

be dilated and no tumour was felt, but there was deep tenderness in the epigastric region.

The diagnosis seemed obvious and operation was indicated.

On October 31, 1904, I exposed the stomach and found on its anterior surface, midway between the pylorus and cardia, a whitish, hard, puckered area spreading up to the lesser curvature and on to the posterior surface of the stomach (saddle-shaped ulcer). The posterior surface of the stomach was adherent to the pancreas. The pylorus was patent and the stomach not dilated.

Posterior gastro-enterostomy was impossible; I therefore performed an anterior operation by suture making a two-inch anastomotic opening in the lowest part of the stomach close to the greater curvature. I brought the jejunum to the stomach after Brenner's method through an opening made in the transverse meso-colon and great omentum, and made the communication close to the duodeno-jejunal junction, no loop being left on the proximal side of the anastomosis.

His after-progress was uneventful. He lost all his symptoms, took food ravenously and rapidly put on weight; on his discharge he was eating ordinary food and expressed himself as being quite well.

I saw him in June, 1905; at this time he was doing his work as a porter and had had no return of his symptoms. In January, 1906, he wrote to me saying: "I am quite well and think the operation successful." I then lost sight of him owing to his change of address. On December 15, 1906, he came to my out-patients' at the London Hospital complaining of pains in the head and "low" feelings. He said he had remained quite well until fourteen days before coming to see me. He had had no digestive troubles of any kind and was taking ordinary food. He was out of work and melancholic, his mental condition bordering on the insane, so that I endeavoured to keep him under observation with a view to the necessary treatment; but this I was unable to do, and was therefore not surprised to find on my visit to Poplar Hospital on February 18 that he had been admitted the previous day, having cut his throat. He died, and the *post-mortem* examination was made the following day.

On opening the abdomen it was difficult to realise that he had had any severe abdominal lesion. There were no visible adhesions and the stomach presented a normal appearance on superficial examination. On looking more closely the portion of jejunum anastomosed to the stomach was almost

embedded in the great omentum, thus rendering its detection difficult and the portion of the stomach to which the anastomosis had been made had become its lowest part. On opening the stomach the fingers at once slipped into the anastomotic opening and it was difficult to pass to the pylorus.

The whole stomach and duodenum were removed together with the pancreas. On opening the stomach widely I found at first no trace of the large ulcer that had been present twenty-eight months before. On more careful search there were traces of scarring on the anterior wall seen on holding this portion of the organ to the light; on the posterior surface, on separating the folds of mucous membrane, the typical puckered scar was seen adherent to the pancreas over an area about the size of a sixpenny piece. This was depressed below the level of the surrounding mucous membrane, but there was no loss of continuity. The anastomotic opening admitted two gloved fingers and the mucous membrane of the stomach was smoothly continuous with that of the jejunum; the pylorus was patent.

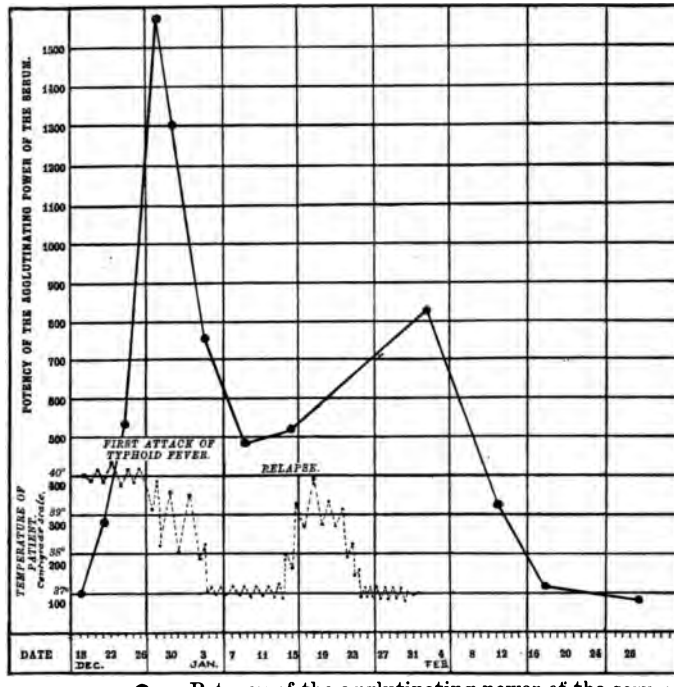
Microscopic sections of the scar showed that the ulcer had healed completely. There was an increase of fibrous tissue in the submucous coat and the muscular tissue was irregular and thinned, drawn up towards the mucous coat and infiltrated with fibrous tissue. There were several microscopic cysts in the deeper layers of the submucosa. The mucous membrane was thinner than normal and the gland tubules irregular.

This case illustrates several important points in the surgery of gastric ulcer. It demonstrates the excellent result obtained by gastro-enterostomy even when the ulcer is on the lesser curvature free from the pylorus and not causing obstruction; also that the anastomotic opening may remain perfectly patent for over two years in the absence of any pyloric obstruction, and confirms the view held by most English surgeons that gastro-enterostomy and not excision is the operation of choice even in chronic gastric ulcers situated elsewhere than the pylorus.

XXX.—*Upon the Extent to which Widal's Reaction persists after Recovery from Typhoid Fever.* By Herbert French, M.A., M.D.(Oxon), and Maurice G. Louisson, M.B. *Read May* 10, 1907.

Introduction.—It not infrequently happens that hospital patients present themselves with symptoms which suggest the possibility of typhoid fever, and yet give a history of having had typhoid fever or some febrile attack which was thought to be typhoid fever previously. The question at once arises whether a positive Widal's reaction in such cases favours the diagnosis of typhoid fever for their present trouble, or whether it may not be due to the former attack. The statements upon this subject inmost text-books are very indefinite; sometimes the teaching is (12) to the effect that the Widal test may remain positive for months and years after the typhoid fever; in other books (1) that the reaction gradually diminishes in intensity until, after a few years, it disappears entirely.

The work of others.—There seem to be few statistics upon the matter. Widal himself (13) repeated his test in seven cases one year after typhoid fever, and found the reaction negative in six, positive in one only. Laming Evans (3) and Jörgensen (6) both worked rather from the point of view of comparing the bactericidal with the agglutinative power of the serum than from that of observing how long it was possible to get a positive Widal's reaction after the fever was past. Evans, using the sedimentation method, repeated the Widal test in twenty-two patients at intervals of two to fourteen months after typhoid fever, and found it positive in five. Jörgensen, on the other hand, found that the agglutinative power of the serum diminished very rapidly; it would rise again during a relapse, but when the typhoid fever was over the reaction became quite negative in a very few months. He expresses his results in the form of diagrams, the following being a typical example:



——●—— = Potency of the agglutinating power of the serum.

·····●····· = The temperature of the patient.

At the end of this paper will be found references to articles by other observers who have repeated the Widal test in small numbers of patients, but nowhere have we been able to find any statistics covering a considerable number of consecutive cases.

The scope of the present work.—Wishing to find out what was the likelihood of a Widal reaction persisting for months or years after typhoid fever, we made a list of all the patients who had given positive reactions when ill with typhoid fever in Guy's Hospital; wrote to each patient, traced as many as we could, sent for these, or went to see them, and repeated the Widal test upon them in the usual way. Our difficulty has been to trace the patients; a great many hospital cases change their addresses frequently, and cannot be followed; out of 281 we have been fortunate in finding 135 again.

The blood was taken in a Widal tube, and in each case sent to the Bacteriological Laboratory for examination in the routine way. The method adopted was that of the hanging

drop, with dilutions of the serum 1 in 20 and 1 in 200, as described in detail by Pakes (11). The examining bacteriologist saw none of the patients, and had no knowledge as to whether the typhoid fever was recent or long past. The results, therefore, are exactly comparable to those that would be obtained in patients who were actually ill. In all cases the cultures of typhoid bacilli clumped readily with the serum of patients suffering from typhoid fever in the wards at the same time, and such patients served as controls throughout the research. Our best thanks are due to Dr. Eyre and Dr. Bainbridge both for help in carrying out the serum tests and for valuable suggestions as to special points in the work.

The standard positive Widal's test.—As Pakes (11) clearly showed, a positive Widal's reaction should be defined as a complete clumping of the typhoid bacilli within half an hour when mixed with serum, the dilution being 1 in 200. He showed that out of 326 cases in which the Widal test was tried 121 proved not to be typhoid fever, and not one of these gave the clumping in half an hour with 1 in 200 dilution of the serum. When less dilution is employed clumping may be partial or complete even when the patient has no typhoid fever. With a dilution of 1 in 20, for example, complete clumping within half an hour is not sufficient evidence by itself that the patient has typhoid fever; such reaction is suggestive but is not conclusive. In all our cases the Widal's test during the original typhoid fever was complete, that is to say, the patient's serum at that time clumped the typhoid bacilli in a hanging drop preparation within half an hour when the dilution of the serum was 1 in 200. We have kept to the same standard in repeating the test, every specimen being examined in dilutions of both 1 in 20 and 1 in 200, and the results recorded after the preparations had been standing fifteen minutes and thirty minutes. Expressed as formulæ, the following represent the various degrees of reaction:

(1) *Negative reactions.*

Dilution of serum.	Minutes. 0	15	30	Dilution of serum.	Minutes. 0	15	30
5 %	−	−	−	5 %	−	−	±
0·5%	−	−	−	0·5%	−	−	−

(2) *Partial reactions.*

Dilution of serum.	Minutes. 0	15	30	Dilution of serum.	Minutes. 0	15	30	Dilution of serum.	Minutes. 0	15	30	Dilution of serum.	Minutes. 0	15	30
5 %	−	−	+	5 %	−	±	+	5 %	−	+	+	5 %	−	±	+
0·5%	−	−	−	0·5%	−	−	−	0·5%	−	−	−	0·5%	−	−	±

(3) *Positive reactions.*

Dilution of serum.	Minutes. 0	15	30	Dilution of serum.	Minutes. 0	15	30
5 %	−	+	+	5 %	−	+	+
0·5%	−	−	+	0·5%	−	+	+

The results obtained.—The results obtained are given in a list at the end of the paper, grouped according to the degree of the reaction. The table on page 163 gives a summary of the total 135 cases.

It thus appears that although in the great majority the reaction becomes entirely negative after recovery from typhoid fever, a positive Widal's reaction may persist in 7·5 cases out of 100, whilst in 22·5 other cases there is a partial persistence of the reaction.

The question at once arises whether the persistence is only for a short time after the fever, or whether it may go on indefinitely. In the following table the cases are arranged according to the time that had elapsed between the typhoid fever and the repetition of the Widal's test:

Table giving the percentages of Negative, Partial, and Positive Widal Reactions at various intervals after the original Typhoid Fever and Positive Reaction.

Interval between the original positive Widal's reaction and the time of its repetition.	Negative reactions.		Partial reactions.		Positive reactions.		Total No. of cases.
	No. of cases.	Percentage.	No. of cases.	Percentage.	No. of cases.	Percentage.	
Between 1 month and 3 months	2	40	3	60	0	0	5
„ 3 months and 6 „	4	67	2	33	0	0	6
„ 6 „ 1 year	3	100	0	0	0	0	3
„ 1 year and 2 years	14	78	4	22	0	0	18
„ 2 years 3 „	13	81	0	0	3	19	16
„ 3 „ 4 „	7	100	0	0	0	0	7
„ 4 „ 5 „	18	86	2	10	1	4	21
„ 5 „ 6 „	12	67	5	28	1	5	18
„ 6 „ 7 „	6	40	8	53	1	7	15
„ 7 „ 8 „	7	54	2	15	4	31	13
„ 8 „ 9 „	4	50	3	38	1	12	8
„ 9 „ 10 „	4	80	1	20	0	0	5

It will be seen that time has very little influence upon it. Apparently if the serum is going to retain its agglutinative power at all it may do so for many years. In the above

Table summarising the number of Cases in each Group, irrespective of the time that has elapsed between the original Typhoid Fever and the repetition of the Widal Test.

Dilution of serum.	Group A.	Group B.	Group C.	Group D.	Group E.	Group F.	Group G.	Group H.
	Minutes. 0 15 30	Minutes. 0 15 30	Minutes. 0 15 30	Minutes. 0 15 30	Minutes. 0 15 30	Minutes. 0 15 30	Minutes. 0 15 30	Minutes. 0 15 30
5 %	− − −	− − ±	− − +	− + +	− ± +	− ± +	− + +	− + +
0·5%	− − −	− − −	− − −	− − −	− − ±	− − +	− ± +	− + +
Total number in each group (grand total 135)	81	13	14	10	6	7	3	1
Percentage of the total 135 in each group	60%	10%	10%	7·5%	5%	5%	2·2%	0·3%
	Reaction negative 70 per cent.		Reaction partial in various degree 22·5 per cent.			Reaction positive 7·5 per cent.		

table it is obvious that the figures to be dealt with are far too small for any but the broadest conclusions to be drawn from them, but it is clear that the Widal's reaction may in many cases disappear in a very short time, and yet in a few may persist for well over eight years at least. The test has not been in vogue long enough for cases to have been followed up for periods of greater length than this.

Another question which arises is whether the age of the patient at the time of the typhoid fever has any effect upon the persistence of the Widal reaction. In the following table our cases are grouped according to age, and it is quite clear that the Widal reaction is no more likely to persist when the typhoid fever occurs at one age than it is at any other:

Table of Cases grouped according to the Age of each Patient at the time of the Typhoid Fever.

Age of patient at the time of the typhoid fever.	Negative reactions now.		Partial reactions now.		Positive reactions now.		No. of cases.
	No. of cases.	Percentage.	No. of cases.	Percentage.	No. of cases.	Percentage.	
5 years old or under .	5	72	1	14	1	14	7
Between 5 and 10 years .	25	76	6	18	2	6	33
„ 10 and 15 „ .	19	83	3	13	1	4	23
„ 15 and 20 „ .	20	67	8	27	2	6	30
„ 20 and 30 „ .	13	59	6	27	3	14	22
„ 30 and 40 „ .	11	58	6	31	2	11	19
„ 40 and 50 „ .	1	100	0	0	0	0	1

When we investigate the influence of sex, however, we find that females appear much less likely to have a persistent Widal's reaction than do males:

Table in which the Cases are divided according to Sex.

	Reaction negative.		Reaction partial.		Reaction positive.		Total No. of cases.
	No.	Percentage.	No.	Percentage.	N .	Percentage.	
Males .	59	65	21	23	11	12	91
Females .	35	80	9	20	0	0	44

It is true that the total number of cases should be much larger than our 135 to make the point absolutely certain. It is, we think, very unlikely that females never exhibit a persistent serum reaction, but seeing that 11 per cent. of our male, and not one of our female patients had a persistent positive Widal's test, the above figures are probably more than mere coincidence.

In attempting to find some cause for the persistence of the positive reaction in those cases who had it, we made notes before the blood was tested of any prominent complications or sequelæ that were attributed by the patients to their typhoid fever. In the list at the end of the paper these are noted; when no note is made it implies that the patient had had ordinary enterica without sequelæ. We have picked out the cases in which some suppurative lesion followed the fever, and tabulated them as follows:

The Influence of Persistent Post-typhoid Suppuration upon Persistence of the Positive Widal's Reaction.

	Case No.		
Widal's reaction now negative	19	Post-typhoidal otorrhœa still discharging right ear	*i.e.* out of 94 cases 3 had persistent suppuration, = 3%
	46	Post-typhoidal otorrhœa quiescent for 8 months	
	85	Purulent discharge from right nostril ever since typhoid	
Widal's reaction now partial		No case of persistent suppurating post-typhoidal	0
Widal's reaction still positive	125	Post-typhoidal empyema discharging for years	*i.e.* out of 11 cases 3 had persistent suppuration, = 27%
	128	Post-typhoidal otorrhœa still discharging both ears	
	130	Post-typhoidal otorrhœa still discharging both ears	

It will be seen that, although post-typhoidal suppuration such as otitis media or empyema seems to render the patient rather more liable to a persistent Widal reaction, the latter may persist without any such cause; and as far as we have been able to judge there is no clinical means of picking out those cases in which the Widal reaction will persist from those in which it will not. This agrees with the views of

Jörgenson (6) and others, namely, that the activity of the serum reaction bears no direct relation to the severity of the disease.

Conclusions.

Our conclusions are:

(1) That in most cases the Widal's reaction disappears rapidly after the typhoid fever.

(2) That in 7·5 per cent. of our cases it remains positive, and may remain so for over eight years, and we do not know how much longer.

(3) That the age of the patient at the time of the typhoid fever has little or no influence on the likelihood of this persistence of the Widal's reaction.

(4) That the Widal's reaction is apparently more liable to persist in males than it is in females.

(5) That the presence of post-typhoidal suppuration somewhat increases the likelihood of this persistence, but is not its only cause.

(6) That the cause of the persistence is not known in most cases; and that it is not possible clinically to say which patient will and which will not give a positive Widal's reaction for years after typhoid fever.

(7) That in any given patient, therefore, in whom the present symptoms might be due to typhoid fever, and in whom there is a possibility of a previous attack, the presence of a complete positive Widal's reaction will leave a 7·5 per cent. doubt as to whether this reaction is due to former enterica rather than to the present illness. The great probability is that a complete positive Widal's reaction indicates present typhoid fever.

Table of Cases in which the Widal Test with Bacillus Typhosus was Repeated Months or Years after the Widal Test was Positive during Typhoid Fever.*

Group A.†—*Cases in which no Reaction occurred even in the Serum Diluted only* 1 *in* 20.

Dilution of serum.	Minutes. 0	15	30
5 %	—	—	—
0·5%	—	—	—

* The cases were all clinically undoubted cases of typhoid fever; by "positive Widal's test" is understood clumping of the *Bacillus typhosus* within half an hour when mixed with serum diluted 1 in 200 (Pakes).

† Groups A and B are "negative reactions"; Groups C, D, and E are "partial reactions": Groups F, G, and H are "positive reactions."

Case No.	Sex of patient.	Age of patient at the time of the typhoid fever.	Interval between original positive Widal reaction and the time of repetition of the Widal test.		Special remarks.
		Years.	Yrs.	Mos.	
1	Male	14	9	9	Had typhoid fever twice, once in 1895, again in 1897. Was in Guy's Hospital with each attack.
2	Female	35	9	5	Married.
3	„	9	9	5	—
4	Male	20	9	2	—
5	„	29	8	11	—
6	„	20	8	4	—
7	„	20	8	1	—
8	„	10	8	0	—
9	„	10	7	10	—
10	Female	20	7	6	Married.
11	Male	16	7	5	—
12	Female	13	7	5	—
13	„	27	7	2	Single.
14	Male	33	6	9	—
15	„	35	6	8	—
16	„	12	6	7	—
17	„	6	6	7	Blepharitis, little or no discharge.
18	„	6	6	3	—
19	Female	5	6	0	Otitis media after typhoid, right ear still discharges.
20	Male	17	5	11	—
21	Female	39	5	10	Married.
22	Male	17	5	10	—
23	Female	12	5	10	—
24	Male	5	5	8	—
25	„	5	5	7	—
26	„	10	5	7	—
27	Female	32	5	7	Single.
28	Male	16	5	4	—
29	„	31	5	3	—
30	Female	11	5	2	—
31	Male	12	5	0	—
32	Female	7	4	11	—
33	Male	19	4	10	—
34	„	11	4	10	—
35	Female	11	4	9	—
36	Male	9	4	9	Brother of 35.
37	Female	26	4	8	Married.
38	„	8	4	8	Has chorea and endocarditis.
39	Male	16	4	7	—
40	„	19	4	6	—
41	Female	8	4	5	Just convalescent from severe scarlatina.
42	Male	12	4	4	—

Case No.	Sex of patient.	Age of patient at the time of the typhoid fever.	Interval between original positive Widal reaction and the time of repetition of the Widal test.		Special remarks.
		Years.	Yrs.	Mos.	
43	Male	7	4	3	—
44	Female	22	4	3	Married.
45	„	31	4	2	Married.
46	Male	18	4	1	Had had otorrhœa for 3½ years, post-typhoidal. It stopped eight months ago.
47	„	18	4	1	—
48	Female	5	4	0	—
49	Male	12	3	11	—
50	„	6	3	11	—
51	Female	18	3	9	Single at time of typhoid fever. Married now.
52	„	31	3	8	Married.
53	„	26	3	7	Pregnant six months when Widal repeated.
54	„	28	3	3	Single.
55	Male	28	3	0	—
56	„	41	3	0	—
57	„	21	3	0	—
58	„	13	3	0	—
59	„	7	3	0	Has many glands in neck, ? tuberculous.
60	Female	14	3	0	—
61	Male	10	2	11	—
62	„	19	2	7	—
63	„	9	2	7	—
64	Female	25	2	6	Single.
65	Male	9	2	3	—
66	„	12	1	10	—
67	„	10	1	9	—
68	„	17	1	8	—
69	Female	28	1	8	Married.
70	„	27	1	5	Married.
71	„	15	1	4	—
72	Male	6	1	4	Has double otitis media, but this dates from scarlatina at two.
73	Female	21	1	4	Single.
74	Male	6	1	3	—
75	„	6	1	2	—
76	„	9	1	0	—
77	„	7	0	7	—
78	Female	5	0	6	"Chest bad and going thin."
79	„	10	0	4	—
80	„	16	0	2	—
81	Male	12	0	2	—

GROUP B.—*Cases in which an indefinite Reaction occurred in thirty minutes with Serum diluted* 1 *in* 20, *but no Reaction occurred with Serum diluted* 1 *in* 200, *nor with* 1 *in* 20 *in less than thirty minutes.*

Dilution of serum.	Minutes. 0	15	30
5 %	—	—	±
0·5%	-	-	—

Case No.	Sex of patient.	Age of patient at the time of the typhoid fever.	Interval between original positive Widal reaction and the time of repetition of the Widal test.	Special remarks.
		Years.	Yrs. Mos.	
82	Male	13	8 1	Has frequent epileptic fits.
83	„	17	7 10	—
84	„	12	6 6	—
85	„	35	4 5	Has had purulent discharge from right nostril ever since typhoid fever.
86	„	12	2 3	—
87	Female	32	2 2	Married.
88	Male	13	2 0	—
89	„	10	1 11	Brother of 66.
90	„	9	1 7	—
91	Female	26	1 2	Married.
92	„	38	0 11	Married. Was pregnant during typhoid. Baby born dead later.
93	„	17	0 5	Single.
94	Male	16	0 4	—

GROUP C.—*Cases in which a definite Reaction occurred in thirty minutes with Serum diluted* 1 *in* 20, *but no Reaction occurred with Serum diluted* 1 *in* 200, *nor with* 1 *in* 20 *in less than thirty minutes.*

Dilution of serum.	Minutes. 0	15	30
5 %	—	—	+
0·5%	—	—	—

Case No.	Sex of patient.	Age of patient at the time of the typhoid fever.	Interval between original positive Widal reaction and the time of repetition of the Widal test.	Special remarks.
		Years.	Yrs. Mos.	
95	Male	13	8 4	—
96	„	25	8 2	—
97	Female	20	7 1	Single.
98	„	31	6 10	Married. Is convalescing from influenza.

Case No.	Sex of patient.	Age of patient at the time of the typhoid fever.	Interval between original positive Widal reaction and the time of repetition of the Widal test.	Special remarks.
		Years.	Yrs. Mos.	
99	Female	24	6 10	Married.
100	Male	26	6 9	—
101	Female	6	6 9	—
102	Male	8	6 0	—
103	Female	22	5 9	Married. Is now pregnant.
104	Male	5	5 4	—
105	„	21	5 2	—
106	„	19	4 5	—
107	Female	18	1 1	Single.
108	Male	18	0 5	Cough ever since. Looks well.

GROUP D.—*Cases in which a definite Reaction occurred in fifteen minutes with Serum diluted* 1 *in* 20, *but no Reaction occurred with Serum diluted* 1 *in* 200.

Dilution of serum.	Minutes. 0	15	30
5 %	—	+	+
0·5%	—	—	—

Case No.	Sex of patient.	Age of patient at the time of the typhoid fever.	Interval between original positive Widal reaction and the time of repetition of the Widal test.	Special remarks.
		Years.	Yrs. Mos.	
109	Male	10	9 3	Has bad acne of face and back.
110	„	9	8 7	—
111	„	16	7 1	Has had bad Malta fever since; also vesical calculus.
112	„	31	6 10	—
113	„	7	6 6	—
114	„	17	6 5	—
115	„	21	6 5	—
116	„	19	5 9	—
117	Female	13	4 3	—
118	Male	32	0 1	—

GROUP E.—*Cases in which an indefinite Reaction occurred with Serum diluted* 1 *in* 200 *in thirty minutes.*

Dilution of serum.	Minutes. 0	15	30
5 %	−	±	+
0·5%	−	−	±

Case No.	Sex of patient.	Age of patient at the time of the typhoid fever.	Interval between original positive Widal reaction and the time of repetition of the Widal test.	Special remarks.
		Years.	Yrs. Mos.	
119	Male	38	1 6	—
120	„	6	1 3	Has a cardiac lesion.
121	Female	34	1 2	Rheumatism and dyspepsia ever since.
122	Male	13	0 4	—
123	Female	17	0 3	Single.
124	Male	37	0 3	—

GROUP F.—*Cases in which a definite Reaction occurred with Serum diluted* 1 *in* 200 *in thirty minutes, but no Reaction occurred with* 1 *in* 200 *in fifteen minutes.*

Dilution of serum.	Minutes. 0	15	30
5 %	−	±	+
0·5%	−	−	+

Case No.	Sex of patient.	Age of patient at the time of the typhoid fever.	Interval between original positive Widal reaction and the time of repetition of the Widal test.	Special remarks.
		Years.	Yrs. Mos.	
125	Male	34	8 1	Empyema followed typhoid. The sinus discharged till quite recently, though to-day it was healed over.
126	„	8	7 9	—
127	„	25	7 8	—
128	„	17	7 5	Double otitis media after typhoid; still discharging occasionally.
129	„	28	6 10	—
130	„	11	5 10	Double otitis media after typhoid. Had mastoid operation in London Hospital nine months ago. Both ears discharge occasionally still.
131	„	7	2 7	—

GROUP G.—*Cases in which a definite Reaction occurred with Serum diluted* 1 *in* 200 *in thirty minutes, and a partial Reaction occurred with* 1 *in* 200 *in fifteen minutes.*

Dilution of serum.	Minutes. 0	15	30
5 %	−	+	+
0·5%	−	±	+

Case No.	Sex of patient.	Age of patient at the time of the typhoid fever	Interval between original positive Widal reaction and the time of repetition of the Widal test.	Special remarks.
		Years.	Yrs. Mos.	
132	Male	24	4 1	—
133	„	16	2 4	—
134	„	33	2 4	Just convalescent from influenza.

GROUP H.—*Case in which a definite Reaction occurred with Serum diluted* 1 *in* 200 *in fifteen minutes.*

Dilution of serum.	Minutes. 0	15	30
5 %	−	+	+
0·5%	−	+	+

Case No.	Sex of patient.	Age of patient at the time of the typhoid fever.	Interval between original positive Widal reaction and the time of repetition of the Widal test.	Special remarks.
		Years.	Yrs. Mos.	
135	Male	3	7 8	—

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XXXI.—*Precocious Obesity, Premature Sexual and Physical Development, and Hirsuties in Relation to Hypernephroma and other Morbid Conditions.*

By LEONARD GUTHRIE, M.D., and W. D'ESTE EMERY, M.D. *Read May* 10, 1906. *With Plates XII and XIII, and Figures.*

BULLOCH and Sequeira in an important paper on "The Relation of the Supra-renal Capsules to the Sexual Organs" (*Path. Soc. Trans.*, April, 1905), have collected a large number of cases of quite young children in whom there has been present a precocious development of hair on the face and on the genital regions, usually associated with precocious development of the genital organs and of the accessory genital glands.

The pathological findings in such cases have been either carcinomata (hypernephromata) or simple hypertrophy of the supra-renal capsules. In a few cases the tumours have been described as sarcomatous in type. These writers showed, moreover, that atrophy and hypoplasia of the supra-renal glands may be associated with disappearance or non-development of hair on the pubes, and hypoplasia of the genital organs. They found that primary blastomata of the supra-renal glands unassociated with precocity are usually lympho-sarcomata, and concluded that the *cortex* of the supra-renal glands is probably connected in some way with the growth of the body and the development of puberty and sexual maturity.

We desire to draw attention to *precocious obesity* as a feature which is at least as striking as precocious puberty, and sexual development in cases of hypernephroma, also to support Parkes Weber's contention that two distinct types of cases are seen in connection with hypernephroma, namely:

(1) The precociously obese type.

(2) The muscular or "infant Hercules" type.

The precociously obese type may be seen in either sex; the muscular type seems to be chiefly met with in males.

We shall also mention cases in which most of, or all the conditions named have been found in association with tumours of other glands than the adrenal, and one in which no tumour of any organ was discovered after death. The true nature of many cases of precocious development remains unexplained owing to absence of *post-mortem* examination.

It is possible that some cases of precocity, sexual and somatic, are to be regarded simply as freaks of nature, or as instances of atavism to a far-off period in which early maturity and propagation were essential to racial existence. The fact that the majority of children who show precocious physical development die young probably gave rise to the saying, "Immodicis brevis est ætas et rara senectus," as applied to precociously intelligent children. In some instances premature development of the sexual and somatic systems may point simply to a condition of "*progeria*," which is incompatible with longevity. The association of precocious development with tumours of the adrenals, of the accessory sexual organs and of the pineal gland suggests that all these organs are in some mysterious way concerned in regulating bodily growth and sexual maturity. Excessive and unbalanced functional activity on their part may possibly induce precocity in one form or another. The coexistence of precocity with tumours in one or other of the various organs named is not invariable. It is only occasional, yet it is valuable evidence of a correlation of function existing between structures whose exact office is obscure.

The relationship between activity of the thyroid and bodily growth, the growth of hair, obesity, and sexual development, is shown in the dwarfing, sparse hair, adiposity and sexual infantilism which accompany myxœdema.

Freund found the thyroid enlarged in forty-five out of fifty pregnant women. It has also frequently been noticed that suppression of menstruation often produces a swelling of the gland, which disappears on the re-establishment of the menstrual flow. J. W. Simpson has traced certain cases of marasmus in infants to deficiency of thyroid secretion ("The Thyroid Gland in Relation to Marasmus," *Scottish Med. and Surg. Journ.*, December, 1906).

One of us has met with a child who menstruated for the first time on her eleventh birthday, and at regular intervals afterwards. Enlargement of the thyroid gland and signs of puberty had been noticed at the age of 8 years, and when seen at twelve years of age she showed a fairly large goitre. The heart's action was rapid (116–120) and forcible. She suffered from palpitation and fainting attacks, but had no other symptoms of Graves' disease.

The thymus gland often shares the enlargement of the thyroid in Graves' disease, thus showing correlation of function between these glands.

Fritsche and Klebs have attempted to refer excessive growth of the body to tumour conditions of the thymus gland (*Ein Beitrag z. Pathologie des Riesenwuchses,* Leipzig, 1884).

Again, the coexistence of gigantism in early cases and of acromegaly in later cases of tumour of the pituitary body is recognised as evidence of the influence of this apparently insignificant organ on osseous growth. The probable relationship between the *Hypophysis cerebri* and hirsuties and sexual functions is illustrated by an interesting case in the clinique of Professor Schloffer, of Innspruck (*Lancet,* June 1, 1907, p. 1528):

A man, æt. 30, suffered from constant headache, anæmia, bitemporal hemianopsia, extreme prostration, and rapid disappearance of all the hair on his body except a few hairs on the chin and the pubes. His sexual power was almost entirely lost.

Examination by X rays revealed the presence of a tumour of the pituitary body. This was removed by operation and proved to be simply an enlargement of the hypophysis. With the exception of the hemianopsia all other symptoms disappeared five weeks after the operation. The man could enjoy life again and his hair began to grow everywhere once more.

It is suggested that hypertrophy of the hypophysis produced over-action of the organ which was detrimental to the growth of hair. We may conclude that a morbid enlargement or tumour of one or other of these organs, namely, the adrenal cortex, accessory genital glands, pituitary, pineal, thymus and thyroid glands, may lead to precocity in one shape or other, either by inducing hyperactivity of function on the part of the organ affected, or by throwing out of gear some restraining or check action which each exerts on the other.

On the other hand, immaturity or cessation of bodily growth and sexual activity have been traced to atrophy or hyperplasia of the adrenal cortex.

Hastings Gilford has recorded a case of progeria or premature senility associated with a shrivelled condition of the supra-renal bodies (*Practitioner,* 1904, lxiii, p. 188).

Wiesel* and Karakaschoff† have referred to cases of hypo-

* "Zur Pathologie des Chromaffen-Systems," *Virchow's Archives,* 1904 Bd. clxxvi, p. 103.

† *Beiträge von Ziegler,* 1904, xxxvi, p. 401.

plasia of the adrenals in females in whom sexual development was immature or its signs disappeared.

Karakaschoff also describes another case running an acute course in a male, æt. 41. It was noted that the testicles were small and the supra-renals were found completely atrophied after death.

Imperfect development of the sexual organs has also been found in association with aplasia or hypoplasia of the adrenals by *Zander* and *Alexander* (*Beiträge von Ziegler*, 1890, vii, p. 439).

The association of imperfect development of the cerebrum and of anencephaly in particular with adrenal aplasia or hypoplasia is well known.

Zander considers the adrenal condition as secondary, whereas *Alexander* regards it as primary to the cerebral defect.

Obesity in males, though not of the precocious type with which we are concerned, but of the Lobengula type, together with gynæcomastia and other feminine characters, are seen in eunuchs, artificial or developmental, whilst in adult females hirsuties and other male characters are associated with ovarian atrophy, and also with hypernephroma.

Mr. John Malcolm removed a large adrenal tumour from a woman, æt. 50, who had had to shave daily for several years (Clinical Society's *Proceedings*, May 10, 1907).

The results of castration on animals need only be mentioned here as evidence of an internal secretion by the sexual organs which exerts a widespread influence on bodily formation in general.

As regards evidence in lower animals of relationship between the adrenals and sexual functions Bulloch and Sequeira say (*op. cit.*, p. 205):

"That there is a possible connection of the supra-renal glands with the sexual system is seen even as low as the frog, when there are seasonal variations in the structure of the gland corresponding to the period of pairing. Apart from the cortical cells and the medullary (chromaffinic) cells of the supra-renal of the "winter" frog, Stilling has shown that in summer considerable changes occur, consisting of a disappearance of the medullary tissue and the development of remarkable cells which from the time of their appearance are called "summer" cells. This is coincident with the pairing of frogs.

Gottschau has also shown in the case of rabbits that the

supra-renal glands undergo changes in volume during pregnancy, the outer zone of the cortex becoming twice the normal thickness at the expense of the medulla and inner zones."

The Precociously Obese Type.

We adduce ten examples of the *precociously obese type.*

Of these seven were female and three male. In six of the female cases hypernephroma was found. In one no tumour of any organ was discovered after death.

Hypernephroma was present in two out of three male cases. In one no *post-mortem examination* was held.

Eleven cases of the *muscular type* were all male.

In two of these hypernephroma was present.

In six no *post-mortem examination* is recorded.

In two the condition was associated with pineal tumour.

In one a tumour of the testicle was present.

In eight female cases in whom obesity was absent or not noted hypernephroma was found in three (Ritchie's, Orth's, and Dobbertin's).

In three, an ovarian tumour was present (Clement Lucas, Rein, Brohl).

In two the subsequent history of the patients is not forthcoming (Betham Robinson).

By *precocious obesity,* a term first used by Parkes Weber, we mean the obesity commonly seen in middle-aged men and women, and not mere plumpness or excessive adiposity such as may be produced in infancy and childhood by excess of patent foods, or may occur as "a normal abnormality" (Goodhart).

In precocious obesity the features are bloated, the cheeks swell with fatness, the complexion is dusky and congested. The skin may be studded with stellate venules; bluish white striæ resembling the lineæ gravidarum are seen about the abdomen and thighs. The fat is usually distributed evenly throughout the body, but the upper or lower extremities may remain thin. Rolls or pads of fat are situated about the neck, breasts, and flanks; a lipomatous mass or hump is sometimes seen between the shoulders. The mammæ may be large and well developed, the belly is pendulous and bulging.

The patient's expression is commonly dull, sad and careworn, and may be suggestive of idiocy. A few have been feeble-minded or imbecile, and possibly the condition described by Bourneville as "nanisme avec obésité" may also

be associated with hypernephroma, though of this we have no evidence. As a rule, however, these precociously obese children are also mentally acute beyond their years.

The circulation is poor, the heart sounds and pulse may be weak, irregular or intermittent, the extremities are cold and blue. Chilblains and small, slowly-healing ulcers form about the toes and fingers. Acne scrophulosorum is not uncommon.

Bronzing of the skin and pigmentation of the mucous membranes with other symptoms of Addison's disease in cases of cortical hypernephroma have only very rarely been recorded.

When present they can only be referred to involvement of the medullary portion of the adrenals in the cortical neoplasm.*

Hirsuties has been present in all recorded cases of hypernephroma. It was shown in early development of pubic hair. In six of Bulloch's and Sequeira's cases it was found on the face. In one it was noticed in the axilla, in one it was generally distributed.

In cases of lympho-sarcoma of the supra-renals, hirsuties is less frequent than in those of hypernephroma.

Precocious development of the external sexual organs was only seen in three of the cases of the obese type collected by Bulloch and Sequeira, namely, Colcott Fox's, J. Ogle's and their own. Their own was the only case in which menstruation occurred and in which the internal organs of generation were mature. But the patient was a girl, æt. 11, who began to menstruate at nine and three-quarter years; the other children were all much younger.

In children of the obese type true precocity of sexual development, apart from the presence of pubic hair, is not a marked feature.

In the *muscular* type, however, which is chiefly confined to the male sex, true precocious puberty and sexual maturity are always present.

It is possible, however, that cases of true precocious puberty menstruation and maternity unassociated with obesity may be analogous to those of the muscular type in males.

* In adults, however, adrenal tumours may be associated with high arterial tension and with extreme atheroma of the arteries. A case in point has been recorded by Parkes Weber (*Path. Soc. Trans.*, July, 1906). The condition probably depends on hypersecretion of the adrenal medulla, for O. Josué and others have shown that high tension and atheroma can speedily be produced in rabbits by injection of supra-renal extract into the veins.

Von Ziemsen, who quotes many authenticated instances of maternity in children aged from nine to twelve years, says: "Such children are, as a rule, in advance of their age in their whole corporeal development" (*Cyclopædia of the Practice of Medicine*, vol. x, p. 322).

He regards such cases as not pathological but as interesting exceptions to general rules.

Hofacker, of Düsseldorf, however, considers precocious puberty and menstruation as evidence of some underlying pathological condition.

Of sixty-two cases which he collected three had sarcoma of the ovary, three were hydrocephalic, and three were rickety. Five of the series became pregnant between eight to ten years of age (*Centralblatt f. Gynäk.*, 1898, No. 41).

The following is a good example of *precocious obesity and hirsuties associated with hypernephroma.*

Henry M., æt. $4\frac{3}{4}$, was admitted to the Children's Hospital, Paddington Green, on October 8, 1906.

He was born healthy and at full term, and had always been naturally plump, but since an attack of measles in 1904 he had become increasingly stout, and hair had grown all over the face, upper lip, and pubes.

He had had pertussis and a bad attack of scarlatina, for which he was detained sixteen weeks in hospital before contracting measles.

Three months before admission he had had several eclamptic seizures. *Family history* was immaterial.

On admission he presented a striking resemblance to a burly brewer's drayman in miniature (*vide* photo, Fig. 1).

Height 36 in. *Weight* 3 st. 1 lb. 10 oz.

The cheeks were enormous, and of firm consistency, and bright red in colour. The cutaneous venules were dilated. The shoulders, trunk and upper extremities were laden with flabby fat which hung in pendulous folds about the mammæ and flanks, and formed a hump like a lipoma in the cervico-dorsal region. The belly bulged with adipose tissue. The buttocks and lower extremities were not especially fat though muscular; in fact, from the level of the sacrum downwards the appearance was that of a normal child.

Hirsuties.—The hair grew low on the forehead, the eyebrows were bushy and joined each other mesially, the eyelashes were long and thick. Coarse light brown hair covered the dorsal hump, and grew down the back, and was also

Fig. 1.—Photograph of H. M., by Dr. Barker, House-physician Paddington Green Children's Hospital.

present on the pubes. He had an incipient moustache, and downy hair on the cheeks and chin.

Organs of generation were not unduly developed for a child of his years.

Heart, lungs and *urine* were normal.

Mensuration.—

Circumference of chest at mammary level 26 in.
" middle arm $7\frac{1}{2}$ in (R. and L).
" forearm $6\frac{1}{2}$ in.
" thigh $11\frac{3}{4}$ in.
" calves (R). 8, (L). $7\frac{3}{4}$ in.

Intelligence.—A bright, precocious child, given to making shrewd and quaint remarks which showed considerable powers of observation.

Three months after admission to hospital he developed acute tuberculosis, of which he died March 11, 1907.

At the *post-mortem* examination the excessive development of the body was noticed, and the fat on the abdominal walls found to be over an inch thick; the muscular system was well, but not excessively, developed. The cause of death was acute tuberculosis, the thoracic and abdominal organs being thickly studded with tubercles in all stages of development, and the abdominal lymph-glands were greatly enlarged and in an advanced state of caseation. These somewhat obscured the anatomical relations of the parts and rendered the dissection of the back of the abdomen difficult.

Apart from the tuberculous deposits the most obvious abnormality in the abdomen was the presence of a rounded tumour, the size of a large walnut, on the left side and in contact with the upper and anterior surface of the kidney, *i. e.* roughly in the position which an adrenal tumour might be expected to take if it slipped downwards and a little forwards by its own weight. It was covered with a fold of peritoneum and was fairly movable, there being no adhesions to surrounding structures. It is fully described subsequently.

Neither adrenal could be found in spite of a careful search. It was thought possible that one or both might have become converted into tuberculous material and so have escaped observation in the tuberculous deposits at the back of the abdomen, some of which occupied exactly the position of the adrenals. With this idea in view all objects having any resemblance in shape to these bodies or lying in proximity to the upper pole of the kidneys were removed and hardened for section-cutting. It appeared later that they were all

tuberculous material, in most cases lymphatic glands, and none of the sections showed any trace of adrenal tissue. It appears, therefore, that the only adrenal substance present was that which occurred in the tumour itself.

The other organs do not require description, since with the exception of the tubercles, which occurred everywhere. they did not present any macroscopic abnormality. The testes appeared normal in size for a child of this age, and the ductless glands (thyroid, thymus, and pituitary body) were of usual shape and size. There was no tumour in the pineal gland, as has been recorded in some cases.

Fig. 2.—Photograph of section of left kidney and the hypernephroma.

Histological report on the hypernephroma (Fig. 2).—The tumour was about 1½ in. by 1 in. by 1 in., and lay in a fold of peritoneum in front of the upper part of the kidney. It was firm and elastic, suggesting that it was cystic in nature, but this is not the case.

On section it is found to be solid, and the cut surface shows an irregular mottling of orange yellow and chocolate, closely resembling the appearance frequently seen in malignant adrenal tumours.

PLATE XII.

Illustrating Dr. Leonard Guthrie and Dr. W. d'Este Emery's Paper on Precocious Obesity, Premature Sexual and Physical Development, and Hirsuties in Relation to Hypernephroma and other Morbid Conditions.

FIG. 1.— × 80 diameters. Section showing the undegenerated portion of the tumour (to the left) and a degenerated area (to the right). *a* is a fully formed alveolus; *b* shows the vascular sinus in which the alveoli lie; *c c* are alveoli with central degeneration; *d* is completely degenerated.

FIG. 2.— × 200 diameters. Showing the alveoli (undegenerated) under a higher power. The endothelial lining of the vascular sinuses is shown at *a*.

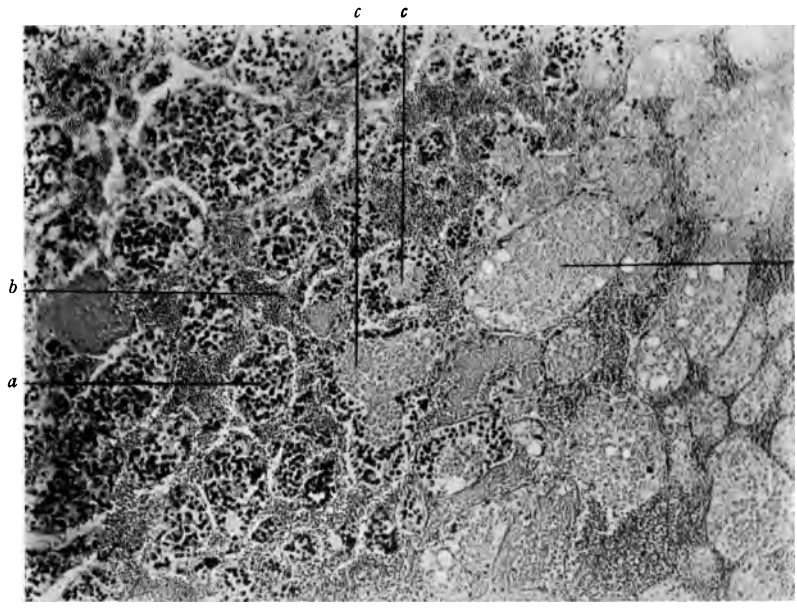

FIG. I

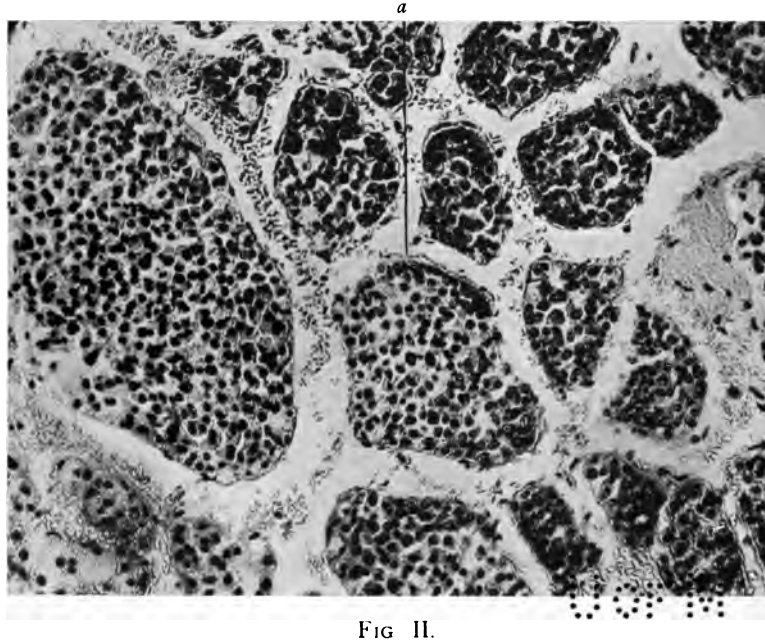

FIG II.

Leonardson & Co.

U of M

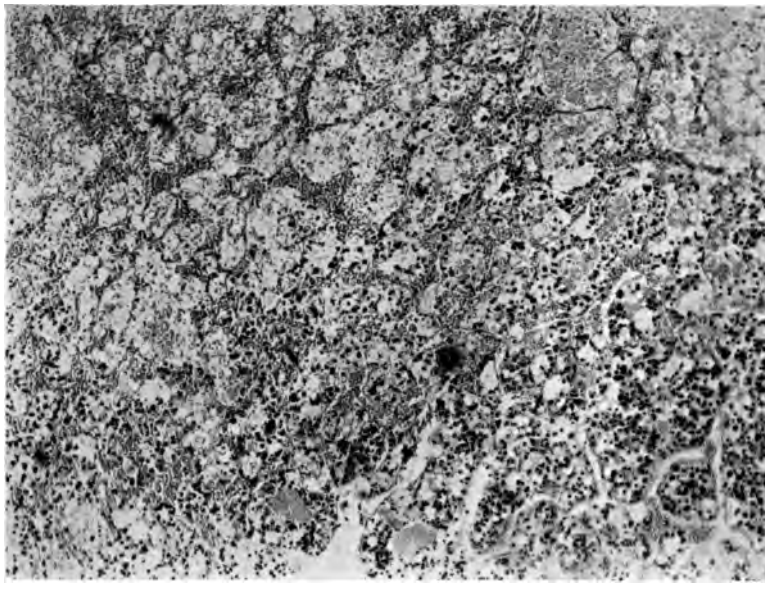

FIG. I.

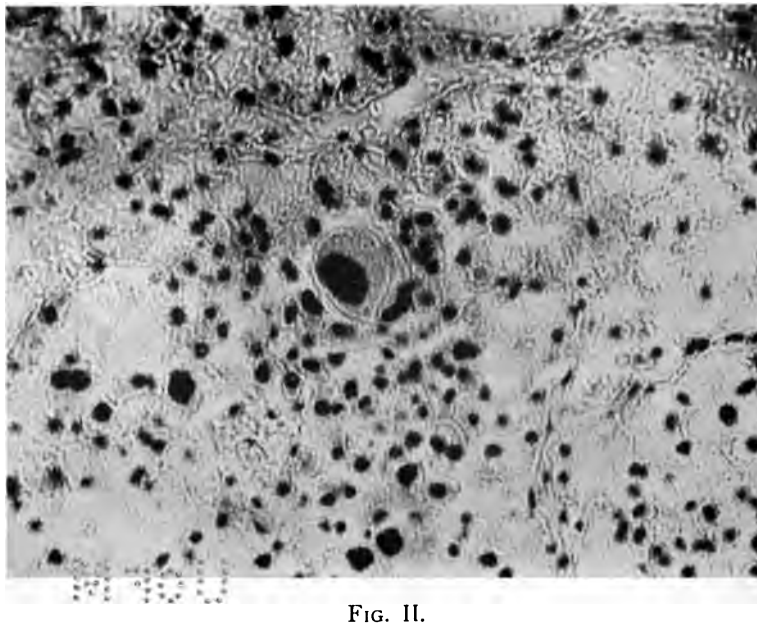

FIG. II.

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PLATE XIII.

Illustrating Dr. Leonard Guthrie and Dr. W. d'Este Emery's Paper on Precocious Obesity, Premature Sexual and Physical Development, and Hirsuties in Relation to Hypernephroma and other Morbid Conditions.

FIG. 1.— × 80. Showing the second form of degeneration described in the text. The right hand lower corner shows undegenerated tumour formation; the left hand upper corner is completely degenerated, and the central zone shows the transition between the two.

FIG. 2.— × 300. Showing a "parenchyma giant cell," about 35 μ in diameter.

The tumour was hardened by Jores' method and not cut into until quite firm. A thin slice was cut from the equator, imbedded in paraffin and complete sections cut. These have been stained by various methods, the following report being based on one stained by hæmatoxylin, followed by picric acid and acid fuchsin (van Gieson's method).

On glancing over the section with a low power it is found that less than half of the tumour is cellular, the majority being composed of necrobiotic tissue (stained yellowish pink) which is in part structureless, though in some places the outlines of the cells, fibres, etc., can be made out; they are entirely unstained by the hæmatoxylin, just as in caseation and in other forms of necrobiosis. Some of these dead areas (readily recognised by the naked eye) are almost ¼ in. in diameter.

There are also large collections of blood which have no very definite walls and impinge for the most part directly on the walls of the alveoli (to be described subsequently) or on degenerated masses of necrobiotic tissue: in some places traces of an endothelial lining is present. The histogenesis of these spaces will be dealt with subsequently. Lastly, there are some small bands of more or less healthy fibrous tissue.

The tumour is bounded by a thin layer of fibrous (peritoneal) tissue which has mostly lost its endothelial coat, though here and there traces are visible.

On turning to the cellular elements we find them collected into alveoli (Pl. XII, fig. 1 *a*, and Pl. XIII, fig. 2) which vary considerably in size, the average being about 80–200 μ in diameter, though larger and smaller forms occur, and in those alveoli that show degeneration the size may be very greatly increased (Pl. XII, fig. 1 *d*). They are roughly circular or polygonal (from flattening by mutual contact) in shape, there being no indication of the existence of solid rods or tubules. These alveoli are mostly separated from one another by a thick column of blood, which forms a network pervading the whole of the cellular portion of the tumour (Pl. XII, fig. 1 *b*, fig. 2, and Pl. XIII, fig. 1). It is bounded by the cellular walls of the alveoli themselves, with here and there an indication of the existence of a partial endothelial coat (Pl. XII, fig. 2 *a*).

The cells themselves are extremely variable. The form most frequently met with is of medium size (about 12–15 μ in diameter) with a circular homogeneous and deeply-staining nucleus and homogeneous protoplasm. Some of the smaller alveoli show this form of cell almost exclusively, the cells being close-set, but in the larger (older?) alveoli there

are other forms, the most noteworthy being "parenchyma giant cells" of very large size (50 μ and upwards in diameter) having a single deeply-staining nucleus. The presence of these cells is of importance, since they are not often met with except in malignant tumours (Pl. XIII, fig. 2).

In many of the cells, especially those which are slightly larger than those described above as the most characteristic form, the protoplasm shows the foamy appearance seen in the cortical cells of the adrenals and in adrenal tumours. The cells as a whole have a decided resemblance to cortical adrenal cells which have undergone great alterations in size and have commenced to degenerate.

Two forms of degeneration of these parenchyma cells can be traced. They occur in different areas of the section, large tracts showing the one form exclusively, others showing only the other.

In the one (see Pl. XIII, fig. 1) the cells in the alveoli swell up (probably from fatty degeneration of the protoplasm), become vacuolated, and lose their power of retaining basic stains. In the section (from which the fat has been removed) they appear as a loose network, in the meshes of which the nuclei can still be seen. As a result (in all probability) of the swelling of the alveoli the blood is squeezed out of the network of sinuses which surround them in the normal state, and large areas of the tumour are now completely extra-vascular. Ultimately the nuclei also degenerate, and the whole tissue is converted into an almost structureless mass.

The second form of degeneration is quite different and its final result is also different (Pl. XII, fig. 1). It commences in the centres of the alveoli, and does not affect the alveolus as a whole, as in the former case. The cells simply undergo necrobiosis without any previous change, and the first effect is the formation of an alveolus with a central area which has lost its power of staining with basic dyes (Pl. XII, fig. 1*c*). This process extends, the alveolus apparently increasing in size until the whole has become necrobiotic. The endothelial coat of the alveolus persists longest, but after a time it also disappears, and the result is the uniform pink staining area described above. In this form the blood is not squeezed out, and can be traced well into the dead patch, where it apparently becomes depigmented and necrobiotic also.

The large areas of blood appear to have originated as follows: The scanty fibrous trabeculæ have in them blood sinuses which are apparently devoid of any walls. This

fibrous tissue swells up and undergoes degeneration, and then the blood seems to escape and form large collections in the degenerated tissue or it breaks through into the surrounding parts.

It will be seen from the above that the tumour is one of very great complexity, and that its nature is not altogether clear. The variation in the size of the alveoli and the presence of parenchyma giant-cells in large numbers undoubtedly suggests the possibility of its being malignant, *i. e.* a carcinoma. Against this must be set the fact that it must have been present for quite a long time (if, as seems reasonable, we are to regard it as the cause of the error in development), but there is no indication of infiltration of the surrounding tissues or of metastasis. It also differs in several respects from the common renal tumours derived from supra-renal rests, notably in the absence of an appearance suggesting that the cells are budded off from the perithelial cells.

The ordinary adrenal adenomata are somewhat similar in appearance to some parts of this tumour, *i. e.* to those in which there is no appearance of degeneration, but in them the alveoli are, as a rule, much more uniform in shape and size and the giant cells are absent. It seems, however, quite possible that these irregularities may be due to the degenerative processes which have obscured the real structure of the parts. The cause of the degeneration is beyond conjecture.

The histology of the other organs does not call for much description, since there was no indication of abnormality beyond the presence of an excessive amount of tuberculous deposit in all stages. The testicles were of a size and degree of development normal to a boy of this age, and there were no spermatozoa to be seen in sections of the testis and epididymis, nor in fluid removed from the vesiculæ seminales. The ductless glands (pituitary body, thyroid and thymus) were also examined and showed no developmental abnormality. The liver, kidneys, lungs, and spleen were extensively affected with tubercle, but were not otherwise abnormal. Sections were also cut of the skeletal muscles and adipose tissue, but showed nothing worthy of remark.

Other Cases of Obese Type in which Hypernephromata have been found.

Bulloch's and Sequeira's patient was a girl, æt. 11. She resembled a stout little woman of about forty years of age.

The breasts were large like those of a sexually mature woman. The pubic regions and axillæ were covered with long hair, and there was a copious development of hair on the chin and lips. Menstruation began at the age of 9¾ years. After death a malignant hypernephroma of the left adrenal body was found. The uterus was much larger than normal; the cervix and body were of equal length, the ovaries a little larger than usual and contained several corporæ lutea, one being of recent date.

Colcott Fox's case was that of a girl, æt. 2. She had been enormous at the age of 10 months, and at 2 years she was somewhat rickety, gross and bloated. The surface of the body was quite remarkably hairy, especially about the genital and paragenital regions. The vulva and clitoris were the seat of precocious development.

After death a large-celled sarcoma was discovered in the left adrenal body. The right supra-renal, as in our own case, could not be discovered. The internal organs of generation are not mentioned.

J. Ogle's patient was a girl, æt. 3. She was enormously stout, weighing 44 lb. She was 2 ft. 10½ in. in height. The girth of the abdomen was 2 ft. 3 in. There was a large amount of hair over the whole body. The eyebrows were excessively thick and bushy. There was a decided moustache, and a large amount of bushy hair on the genital organs, which were well developed.

Post-mortem.—An "encephaloid cancer" was found in the left adrenal gland. The condition of the internal organs of generation is not stated.

Linser quotes Tilesius (1803) as having observed in a girl, æt. 4, who measured 80 cm., an enormous formation of adipose tissue, with premature development of the mammæ and the pubic hair.

The autopsy revealed a tumour the size of a goose's egg in the site of the left supra-renal capsule with metastases in the liver.

William Cooke (*Phil. Trans.*, 1756) described a girl, æt. 7, who was enormously fat and had a copious growth of hair on the face and genitals. There was a large tumour in the site of the left supra-renal body.

Bevern and Romkild (*Neues Journal d. prakt. Heilkunde*, 1802) reported the case of a girl, æt. 3½, who looked like a woman of twenty. She was very fat, had abundant hair on her genitals, also on her face. The necropsy showed a large

tumour connected below with the uterus, but above with a large glandular body in the region of the left curvature of the colon and the spleen.

Precocious Obesity and Hirsuties unassociated with Hypernephroma.

Ethel W., æt. $2\frac{9}{12}$, was admitted to Paddington Green Children's Hospital, December 7th, 1905.

She had always been a rather plump child, but for three months she had grown rapidly fatter. She complained of pains about the head and face, had "no life in her," slept a great deal, and was always blue and cold.

She had had measles, whooping cough and chickenpox, but not scarlatina.

Family history was unimportant.

The photograph (Fig. 3) taken six months later suggests that of a little stout woman at about the climacteric period.

Height 2 ft. 9 in; weight 37½ lb.

She had grown 1 in. and increased 6 lb. in weight in three months.

The features looked small and the temporal regions were masked by the cheeks, which were distended with fat, dusky red, and blotched by distended venules.

Pads or rolls of fat existed round the neck, chest and flanks, and a well-defined hump or lipoma was present in the cervico-dorsal region.

The fat was otherwise evenly distributed, and affected the lower as well as upper extremities. In the folds of the groins and abdomen were numerous purplish white striæ resembling "lineæ gravidarum." The breasts were large and pendulous.

Hirsuties.—Hair on the head was fairly thick and abundant. None was present on the face or in the axillæ, but the labia majora was covered with coarse brown hairs an inch in length.

The external organs of generation were infantile. Menstruation had not occurred, but there was an offensive discharge from the vulva.

Measurements.—Round neck, 12½ in.; chest, 25¼ in.; abdomen, 26½ in.; upper arm, 8 in.; lower arm, 7½ in.; thighs, 13¾ in.; calves, 8¼ in.

Fig. 3.—Photograph of E. W., by Dr. Attenborough, House-surgeon Paddington Green Children's Hospital.

The circulation was poor; the heart sounds weak, irregular and intermittent. Pulse rate 120–130 per minute.

The temperature averaged 97°–98°F.

Urine was normal.

The expression was usually dull and sad as shown in the photograph, but became animated when the child was amused or teased. She was precocious in intelligence, and, like the child H.M., when irritated her language was more voluble than refined.

In the early part of October, 1906, a large abscess formed in the left groin. This was opened, and the wound had nearly healed when she was seized with uncontrollable vomiting and diarrhœa, of which she died.

Copy of the post-mortem notes. "*External examination.*—Extreme degree of general adiposity, development of pubic hair. *Abdomen.*—No free fluid. *Supra-renal glands* are healthy. *Uterus* is infantile, as also are the *ovaries*. Other organs present nothing abnormal. *Thyroid gland* is present, no atrophy. *Heart* and *lungs* are normal. *Brain.*—No abscess or tumour, nothing visible to naked eye."

This case resembles in every particular the precociously obese type associated with tumour of the adrenals. It is to be regretted that no report of microscopical examination of the organs is forthcoming.

The patient during life was exhibited at the Clinical Society's Meeting, April 27, 1906, and also at a Meeting of the Society for the Study of Disease in Children (*Reports*, vol. vi, p. 146).

Precocious Obesity and Hirsuties; (?) Hypernephroma or (?) Tumour of Pituitary or Pineal Gland.

Dr. Still (*Clin. Soc. Trans.*, vol. xxxiv, 1901, p. 243) has published a case of unusual obesity in a boy, æt. 7¼.

A year previously he had been very thin, he then rapidly became immensely fat. His height was 4 ft. 2½ in., his weight 4 st. 11½ lb.

For two months he had been short of breath, and a month before he came under observation he had suddenly become blind for forty-eight hours. Occasionally he suffered from pains and sensation of pins and needles over the vertex of the head.

"His general appearance was striking owing to great accumulation of subcutaneous fat; his cheeks were red and bulging, and over the rest of the body there was a great excess of subcutaneous fat in all parts where it is not usually present. In the supra-clavicular fossæ and over the *vertebra prominens* behind, the collections of fat were perhaps more marked than elsewhere, but otherwise the increase of fat was uniform and diffuse."

The abdomen was large, its prominence being due chiefly to fat in its walls. The lower part of the abdomen and upper part of thighs showed irregular bluish red lines where the skin was thin and shiny with dilated venules evidently analogous to the lineæ gravidarum.

There was some hair over the pubes said to have been present for two years. The voice was not gruff; the right testicle was normal, the left was not felt. The thyroid gland was certainly not enlarged. The heart was slightly irregular in rhythm, the second sound was markedly accentuated.

He died, Dr. Still informs us, a few months later, with cerebral symptoms suggesting hæmorrhage. A *post-mortem* examination was not made, but this description of the case leaves little room for doubt that it was of the nature of those under consideration.

Cases of Hypernephroma with Precocious Hirsuties but without Obesity.

Orth has described a girl, æt. $4\frac{1}{2}$, in whose abdomen a tumour had been noticed since her second year. Hair on the pubes had been noted for nine months, and for six months the beard had had to be shaved. The external and sexual organs were precociously developed, the clitoris being like a small penis. Menstruation is not mentioned nor the presence of obesity. After death carcinoma of the right adrenal was found. The condition of the internal organs of generation is not described (*Arbeiten aus dem path. Institut zu Göttingen, Berlin*, 1893, p. 75).

Dobbertin met with a girl, æt. $1\frac{2}{12}$, who had fairly thick hair 5 cm. in length on the great labia, lower and lateral abdomen, thorax, back and cheeks.

She was well nourished and normally built, but not apparently obese. After death a hypernephroma was found on

the left side. The condition of the organs of generation is not described (*Beiträg z. Casuistik der Geschwülste, Beiträg z. path. Anatomie v. Ziegler*, 1900, xxviii, p. 42).

Dr. Ritchie, of Oxford, supplied to Drs. Bulloch and Sequeira (*op. cit.*) the account of a girl, æt. 4, who died three days after osteotomy, without any apparent cause except that the wound became slightly infected. At the autopsy all the organs were normal with the exception of one supra-renal gland, which was of the size of a kidney and was the site of a tumour which was in type that of a sarcoma.

The genital organs during life had been thought to be hermaphroditic, but were pronounced by Professor Arthur Thomson to be normal except that they were of the size of organs of a developing woman. There was no history of menstruation, but there existed an abundant development of pubic hair.

Dr. Ritchie kindly informs us that "the child was well nourished but not obese in any sense. Apart from the premature development of the genitals there was no abnormality. One would have said she was merely an ordinary plump girl."

Unilateral Precocious Obesity without Hirsuties, associated with Adrenal Hyperplasia and Hypertrophy of Paired Organs on the left side.

A remarkable instance of this kind has been recorded by Dr. R. Hutchison.

The patient, a boy, æt. 4 months, showed considerable hypertrophy of the left arm and leg. The left side of the trunk was similarly affected but to a less extent.

After death from broncho-pneumonia the autopsy showed the enlargement to be due to increased deposit of fat in the connective tissues. The bones were symmetrical; the brain, pineal and pituitary glands were normal. Most of the paired organs were larger on the left side than on the right. Left kidney weighed 56 grm.; right 28 grm.; left supra-renal 42 grm.; right 14 grm.; left testicle 2·3 grm; right 0·55 grm.

The condition of the enlarged supra-renal appeared to be a hypertrophy and hyperplasia (*British Journal of Children's Diseases*, June, 1904).

Muscular or Herculean Type.

The characteristics seem peculiar to the male sex. We know no instances of their occurrence in females unless those of precocious maternity are analogous. They consist in true precocious development of sexual maturity with hirsuties and remarkable growth of stature and muscular strength—without obesity.

Legends concerning Hercules and Samson may have their origin in the occasional appearance of individuals of this type.

Pliny (*Hist. Nat. Lib.*, vii—xxvii) mentions the son of one Euthymenes, of Salamis, who in three years grew to be 4½ ft. in height, and arrived at the state of puberty. His gait was slow, his wits were dull, his voice great, and at three years of age he died of a general cramp (Anthony White, *Med.-Chir. Trans.*, vol i, 1806-09).

Craterus, the brother of King Antigonus, knew a person who in seven years was infant, youth, adult, parent and old man (*Phlegon de Mirab.*, cap. xxxii).

In the *Philosophical Transactions* for 1744-5, vol. xliii, p. 250, there is an interesting account of the gigantic boy of Willingham, Cantab. A complete history of this case was published in pamphlet form by Thos. Dawkes, surgeon, of St. Ives, in 1747.

He was born on October 31, 1741, and was uncommonly large at birth, especially "in partibus generationis." At the end of his first year "lanugo appeared, which gave great uneasiness to his parents, who were very religious people." Shortly afterwards his mother died suddenly whilst suckling him, and it was presumed that he absorbed her vitality like a vampire.

At $2\frac{10}{12}$ years his height was 3 ft. $8\frac{5}{10}$ in. and he grew 3 inches taller in 3 months. His weight was 4 st. 2 lbs. Hair on the pubes was an inch in length. The penis when pendant measured $3\frac{3}{10}$ in. and "in statu erectionis" 4 in. His voice was "as deep or bass as you can well conceive." He was very strong, and could throw with much facility a blacksmith's hammer weighing 17 lb. He did not fight but collared and threw with ease boys of eight or ten years of age.

He died at the age of 5 years of a disease resembling phthisis pulmonalis, and when dead had the appearance of a venerable old man.

Anthony White also describes the case of Philip Howarth, who was born February 21, 1806.

He was a ninth child, healthy and large when born, and the cranial sutures were closed at birth. At twelve months he could run alone. His features, from being plump and infantile, grew suddenly long, pale and extremely ugly. The unhealthy appearance of boys at fourteen was strikingly marked at one year. His penis and testicles increased in size, black curling hair appeared on the pubes and his voice changed.

At $2\frac{8}{12}$ years he was nearly the exact representation of the "Farnese Hercules." His height was 3 ft. 2 in., his weight 47 lb. His voice was that of a youth of sixteen; his genitals were as large as in some adults. He had no beard, but acne spots or "blackheads" appeared on his face. He masturbated at the end of his second year and voided "semen perfectum et bene elaboratum."

We are unable to trace his history further.

About twenty-five years ago Mr. Bruce Clarke had under his care at St. Bartholomew's Hospital a child, æt. 3, who conformed exactly to the muscular type of precocity. We have not been able to ascertain his subsequent history.

A Modern Instance of such Precocious Sexual and Somatic Development.

Mr. Hugh Lett exhibited before the Society for the Study of Disease in Children (March 16, 1906) a boy, æt. 4 (Fig. 4), who had been rather backward until two years old, when pubic hair appeared, and development took place with great rapidity.

There was a remarkable growth of hair over the whole body, especially over the pubes and in the lumbar region. The penis and testicles were large and resembled those of a young adult. His voice was deep, and was like a bass voice not yet under full control. His muscular development was remarkable, especially in the thighs, and he was possessed of proportionately great physical strength. A skiagram of the wrist showed ossification taking place in all the bones of the carpus, and one of the knees presented the appearance of the joint of a boy seventeen to eighteen years old. No enlargement of the thyroid or supra-renal bodies could be made out.

Intellectually he was bright, but had a terrible temper. He had no fits nor was he subject to headaches. His appetite was voracious.

FIG. 4.—Photograph of Mr. Hugh Lett's case, reproduced by the kind permission of the publishers of the *Reports of the Society for the Study of Disease in Children*, 1906.

There had been nocturnal enuresis, frequent erections and nocturnal emissions during the previous three to four months.

Height 3 ft. 7½ in., weight 3 st. 12 lb.

Cases of the Muscular Type in which Hypernephromata have been found.

Linser's patient was a boy, æt. $5\frac{7}{12}$, who was supposed to be sixteen to eighteen years of age. He exceeded most of the measurements of a normal boy of fifteen. The mons veneris was covered with hair. The penis was 8–9 cm. in length, and 12–14 cm. "in statu erectionis." The testicles were as large as pigeon's eggs.

After death a malignant tumour in the left supra-renal was found ("Ueber die Beziehungen zwischen Nebenneiren und Körperwachstum besonders Riesenwuchs," *Beiträge z. klin. Chirurgie*, 1903, xxxvii, p. 282).

Charles E. Adams, records the case of a boy æt. $14\frac{9}{12}$, who developed puberty at the age of ten years, from which time although he increased little in height he developed great muscular strength, excelled in athletic sports, and defeated all competitors. For two years his complexion had been observed to be plethoric and dusky, and during this period the growth of hair on his face had to be shaved almost daily. His general appearance was that of a sturdy little man.

He died at the age of sixteen of a malignant hypernephroma like a perithelioma affecting the left supra-renal capsule (*Path. Soc. Trans.*, vol. lvi, Pt. II, 1905, p. 209).

Cases of the Muscular Type in which other than Adrenal Tumours have been present.

C. Ogle has recorded the case of a boy, æt. 6, who showed a marked premature development of the penis and the pubic hair. After death a tumour, apparently being an alveolar sarcoma, with hæmorrhages and cysts involving the *pineal gland* was found (*Path. Soc. Trans*, 1899, vol. l, p. 6).

Heubner has observed a similar case of pineal tumour in a boy, æt. $4\frac{1}{2}$, with abnormally large penis, scrotum and testes, and abundant pubic hair (quoted by C. Ogle, *op. cit.*).

Muscular Type associated with Testicular Tumour.

E. Sacchi (*Riv. Ref. di Frenetria*, 1895, vol. xxi, p. 149) describes a boy, æt. $9\frac{1}{2}$, whose development was altogether excessive. His right testicle was atrophied, the left was of enormous size. It was removed and found to contain an epithelial growth. "The boy then became more childlike, and his beard fell out."

Cases in which Precocious Puberty has occurred in association with Ovarian Tumours.

Clement Lucas recorded the case of a red-haired girl, æt. 7, who was admitted to the Evelina Hospital after having menstruated several times. Hair an inch in length grew on the pubes, the mammæ were well developed and the child resembled a developed woman. Mr. Lucas removed a solid tumour of the ovary, and after the operation menstruation ceased, and the signs of puberty gradually disappeared.

The tumour seems to have been of doubtful nature. Dr. Goodhart said, "Round-celled sarcoma seems the best name for it." It consisted of numerous alveoli containing 1–3 granular cells. It was probably not malignant (*Clin. Soc. Trans.* vol. xxi, 1888, p. 224).

Parkes Weber (*Reports of the Society for the Study of Disease in Children*, vol. ii, 1902, p. 232) mentioned that *Rein* detected an ovarian cyst in a girl, æt. 6, who menstruated prematurely (*Vratch*, 1894).

Brohl removed a large cystic ovary from a girl, æt. 7, with precocious menstruation. The child remained under observation for about two years during which there was no further menstruation (*Centralblatt f. Gynäk.*, 1897, No. 4).

Hofacker found three cases of sarcoma of the ovary out of sixty-two cases of precocious menstruation (*Centralblatt f. Gynäk*, 1898, No. 41).

Other unexplained Cases of Precocious Puberty.

H. B. Robinson (*Reports of the Society for the Study of Disease in Children*, vol. ii, 1902, p. 222), referred to an interesting series of cases of precocious sexual development in a single family.

Two children, one a fortnight and the other two years old, were brought to him to decide whether they were male or female. He said that the elder was a female, but was doubtful about the sex of the younger. Eighteen months later they were brought to him again, and then the elder girl had developed a good deal of pubic hair, and the younger child, who was then found to be a female, had also signs of hair on the pubes. In both cases the clitoris was much hypertrophied. Menstruation had not occurred. A curious feature was that

their brother, a child æt. 5, had genitals and pubic hair such as would be seen in an adult.

We know no other instance in which precocious puberty to this extent has occurred in members of the same family.

The Nature of the Tumours concerned in the production of Obesity with Premature Sexual Development.

In the majority of cases it has been described as carcinoma or hypernephroma malignum, but in two (Colcott Fox's and Ritchie's) the structure has been that of a sarcoma; in several others there has been a simple hypertrophy.

In the case (H. M., see p. 180) now published the tumour, if malignant at all, is of a carcinomatous type.

The occurrence of a true carcinoma at this early age is decidedly rare, and a few remarks on some recent views as to the true nature of these tumours may not be out of place. It has long been known that tumours of the adrenals and of some other parts of the body do not conform exactly to the classical types of carcinoma and sarcoma as met with in other organs. In many cases they approach closely to the type of the endotheliomata, and in special resemble the peritheliomata, in others sarcomatous and carcinomatous structures may appear mingled together, and in yet others the primary tumours may be carcinomatous in type and the metastases sarcomatous. Thus Rolleston and Marks say: "Malignant tumours of the adrenals are a peculiar and special class. They may approach structurally either the carcinomata or the sarcomata, and sometimes one and the same tumour may in parts resemble each." *

There is, however, no absolute agreement amongst pathologists as to the use of the terms, and every attempt to define them so as to conform to the use of different authorities has been unsuccessful. There are two main methods of classification—the morphological and the developmental or embryological. If we adopt the former the diagnosis of the nature of any tumour is simple, and it is easy enough to label it sarcoma or carcinoma according to its structure. We find, however, that the groups thus obtained are not at all homogeneous, the carcinomata, for example, including the highly malignant scirrhus and some almost benign endotheliomata. Nor is the embryological or developmental classification

* *Amer. Journ. Med. Sci.*, 1898, p. 383.

altogether satisfactory. It is by no means easy to apply in practice, since the study of the histogenesis of any tumour is a difficult matter. Further, it seems irrational to separate two tumours of similar structure into different groups because of their differing embryology, and this is what we must do if we adopt this classification, since we must regard any malignant adrenal tumour as a sarcoma (because of its origin from mesoblast), thus separating it from other malignant tumours which it closely resembles, but which are derived from epiblastic organs. Yet the embryological classification is on the whole most satisfactory and most generally used, and by pushing our researches in development a little further than the recognition of the three primary layers it is possible to harmonise the embryological and structural views on the adrenals.

The mesoblast separates, soon after it is formed, into two parts, the median mass, or mesenchyme, from which the connective tissues are derived, and the distal portion, which divides into two layers and forms the endothelium lining the cœlom and the uro-genital tract. This is called mesothelium, and it is from this portion that the adrenals, and therefore the adrenal tumours, are developed, hence the name mesothelioma which has been applied by Buxton* and by Woolley and Adami† to the tumours derived from the adrenals, the ovaries and testes, and the kidneys.

This view accounts for the structure of the tumours of these organs in a very satisfactory way. The "epithelium" of these mesothelial organs is not embryologically epithelium at all, being derived from the mesoblast. As a result, if we accept the view that a tumour is, structurally, a reversion to an earlier type, we can readily explain why it should form tumours of epithelial or sarcomatous structure; in the former case the reversion has not proceeded far, whereas in the latter case the cells have gone back to their primary undifferentiated mesoblastic condition. It follows, therefore, that the most logical course is to cease to attempt to classify the tumours of these organs as if they were derived from epithelium or connective tissue, and to call them mesotheliomata, adding for greater exactness (if necessary) "of carcinomatous type," or "sarcomatous type," or "mixed."

In the cases recorded the reversion does not seem to have gone far. The majority of the tumours have been mesothelio-

* Buxton, *Journ. Cutaneous and Genito-Urinary Diseases*, 1890, p. 74.

† Woolley and Adami, *Trans. Assoc. Amer. Phys.*, 1902, xvii, 627.

mata of carcinomatous type, two only being described as sarcomata.

On the other hand, true cases of sarcoma do occur in the adrenals, and appear to be unassociated with signs of precocious development. Thus in the cases described by de Huyter* and Pepper† the tumours were lympho-sarcomata, and in eight other cases collected by Bulloch and Sequeira the sarcomata were present in cases in which the bodily development was normal.

It would appear from these facts that the cause of the precocious development lies in the parenchyma-cells of the adrenal cortex, and is probably of the nature of a hyper-secretion of an internal secretion. When a true sarcoma (*i. e.* one developing from the connective tissue and not from the parenchyma) occurs, it seems probable that no change in the activity of the cortical cells occurs, or that they are destroyed, in which case no internal secretion is formed. When, however, an overgrowth of these cells occurs, whether in the form of simple hypertrophy or of a tumour of sarcomatous or carcinomatous appearance, we must assume that the internal secretion is increased in amount or altered in quality in such a way that acting on the growing tissues, or possibly in virtue of some indirect action on other organs having an internal secretion, it alters their metabolism in some profound way and causes the general changes with which we are now familiar. It is perfectly true that tumour-formation is in general associated with a diminution of the function of the cells concerned, and that we should expect, therefore, a diminution of the internal secretion of the cortical cells when meso-theliomata are present. There are, however, indications that this rule is not without exceptions, and it is difficult to explain the observed facts on any other hypothesis.

Conclusions.

(1) Precocious development, sexual and somatic, may be due to tumours or to simple hypertrophy of the adrenal cortex, of the accessory genital glands, and of the pineal and pituitary glands. The thyroid and thymus glands are also probably intimately connected with the phenomena of physical and sexual development.

(2) The opposite condition, namely infantilism, stunted

* *Archiv für klin. Chirurg.*, 1890, p. 98.

† *Amer. Journ. Med. Science*, 1901, p. 287.

growth, loss or non-development of sexual function may depend on atrophy, or on perverted or annulled function of one or more of the organs named.

(3) Hypersecretion on the part of one or more of the glandular structures named in the first case, and hyposecretion in the second, may account for the opposite conditions described.

Correlation of function or some mutual check action may exist between the various organs which have been mentioned.

(4) Precocious development, sexual and somatic, may in some cases be unassociated with any obvious lesion of organs.

(5) Two distinct types of precocious development, namely the *obese* and the *muscular* type, are found in association with tumours of the adrenal cortex. The obese type affects both sexes, but the muscular is practically confined to males.

(6) Premature hirsuties is the rule in practically all cases of premature development, but is not necessarily associated with other signs of sexual maturity

(7) The nature of adrenal tumours which have been associated with precocious development is, as a rule, such as would suggest an overgrowth of the parenchyma-cells proper to the adrenal, and may be either malignant or non-malignant; whilst in some, at least, of the cases of hypernephroma which are not associated with this precocious development, the structure (*e. g.* that of a lympho-sarcoma) would suggest destruction of these cells.

XXXII.—*Diagnosis in Cases of Prolonged Pyrexia.*

By F. DE HAVILLAND HALL, M.D. *Read May* 24, 1907.

THE occurrence of two cases of prolonged pyrexia of the intermittent or hectic type under my care in the Westminster Hospital has induced me to discuss the difficulties which may arise in arriving at a correct diagnosis in similar cases. I will briefly narrate the clinical history and *post-mortem* appearances of the two cases.

CASE 1.—R. B., æt. 44, meat-carver, admitted June 10, 1901. Patient was admitted suffering from extreme weakness. Father died, æt. 84, of cancer in neck. Mother, æt. 76, alive and healthy. Two sisters and three brothers alive and healthy. Four brothers and sisters died young.

Patient was born in London, and except for five years when he was in the Isle of Wight, he has always lived in London. Six years ago he attended the Out-patient Department suffering from rheumatism. He had gonorrhœa when eighteen years of age, but has not had syphilis. In July, 1900, he had "congested liver," after treatment at home he attended St. Bartholomew's till February when he was too ill to go. He then had influenza. Whilst attending at St. Bartholomew's he was found to have some "tuberculous glands" in the left side of the abdomen. At the end of February patient improved, the glands cleared up, and at Easter he felt fairly well. He then caught cold, and the cough returned.

For about three weeks before admission he had been attending the Out-patient Department. On June 7 he brought up about a tablespoonful of blood. He has lost weight.

On physical examination the only thing found amiss was a patch of impaired resonance and deficient entry of air at the right apex posteriorly, a few moist sounds were heard in the interscapular region. The heart-sounds were quite normal.

In view of the temperature, loss of weight, history of bringing up blood, coupled with the physical signs at the

right apex a diagnosis of pulmonary tuberculosis was made. There was, however, very little cough, and hardly any expectoration.

On June 21 the sputum was examined and *B. tuberculosis* could not be found.

24th.—It was noted that the physical signs remain unchanged. The patient does not feel so well in himself.

July 1.—Some enlarged glands can be felt in the left inguinal and iliac regions.

13th.—During the past week slight œdema of the feet has been noticed. There is no fluid in the pleural or peritoneal cavities.

19th.—The œdema is more marked in the left foot than in the right. The tongue is moist and slightly furred. The appetite is not so good. Nothing further has been discovered on physical examination.

13th.—Examination of blood:

Red corpuscles (good size and shape)	2,283,000	per c.mm.
Leucocytes	3,520	„
Finely granular oxyphil cells	81	per cent.
Coarsely „ „	—	„
Hyaline cells	8	„
Lymphocytes	11	„
Hæmoglobin value of blood	60	„

Numerous blood platelets.

19th.—4 c.c. blood taken from median basilic vein and added to 50 c.c. broth. No micro-organisms of any kind grew by the end of two days.

22nd.—The scrotum became œdematous yesterday; the œdema of the feet has increased. The glands in the left iliac region have increased in size.

August 12.—Patient's general condition has remained unaltered. Hæmorrhage in both retinæ has been noted. Liq. Arsenicalis ɱiij ter die ordered.

21st.—Diarrhœa commenced on the 15th, and has persisted until now. The liquor arsenicalis has therefore been stopped.

26th.—The diarrhœa is less. The glands in the supraclavicular region appear to be increasing in size; the other glands are unaltered.

September 4.—A soft systolic murmur heard loudest at third left interspace has been detected. It is not audible in the axilla. The impulse is feeble and the heart does not

appear to be enlarged. Œdema of ankles more marked since the patient has been up. The diarrhœa has ceased.

13th.—Patient had 10 c.c. of antistreptococcic serum subcutaneously.

These injections were continued daily for ten days without any appreciable effect, so they were omitted.

During October the patient's condition fluctuated from day to day, but on the whole he gradually grew weaker.

November 8.—Patient had a fit at 6 a.m. It lasted about two minutes. It started in the right arm and then spread to face. The arm remained weak for a few hours. Two days later he had another fit.

On November 26 he had two fits, and one fit on the 28th and 29th respectively.

He died on November 29.

On admission the patient weighed 8 st. 7 lb. 10 oz. in his dressing gown. He used to weigh about 12 st. The weight gradually fell to 8 st. 3 lb. 4 oz. on June 30. From this date there was practically a steady rise in weight until August 25, when the patient was found to weigh 9 st. 3 lb. By September 10 he had lost a stone. From this date the weight fluctuated between 7 st. 12 lb. and 8 st. 3 lb. 10 oz. His last weight was 8 st. 1 lb. on November 2. After this he was too weak to be weighed. Much reliance cannot be placed on the weight, as the amount of dropsy varied.

The *temperature* from the date of admission until October 31 was of a characteristic hectic type, usually about 103° F., and coming down to normal, or nearly normal, in the morning. The highest temperature attained was 103·7°. In the first week of November the temperature was practically normal; it then rose again, but only once reached 102°. For the three days preceding death the temperature was mostly subnormal.

The urine varied in specific gravity from 1016 to 1022. On several occasions in the month of September a trace of albumen was detected; later on the urine was free from albumen. Sugar was never found.

The medicinal treatment was very simple. At first he was ordered a prussic acid and soda mixture; then he took 2 gr. of quinine every four hours for a month. As the temperature was not affected by the quinine, liquor arsenicalis in 3-minim doses was given, but, as this caused diarrhœa, it was discontinued, but resumed later on. From time to time, when he had dyspeptic symptoms, he went back to the prussic acid and soda mixture.

Necropsy made twenty-two hours after death.

Body much emaciated. Slight excess of fluid in all four great serous sacs.

With the exception of sub-arachnoid œdema and excess of fluid in the ventricles the brain is normal.

The pleuræ are normal.

Lungs are large. Some hypostatic engorgement and œdema. At right apex there is consolidation about the size of a walnut; it is quite firm; it is surrounded by a sinuous yellow margin, the central portion being greenish-black, mottled with yellow. Rest of lungs normal.

Heart and arteries normal.

Peritoneum normal.

The stomach at the pyloric end presents four ulcers of the mucosa. Each is about the size of a threepenny piece. A similar ulcer is seen in the sigmoid flexure, oval, about the size of a bean. The rest of the gastro-intestinal tract is normal excepting for atrophy of lymphoid tissue.

The liver and pancreas are normal.

The spleen weighs 13 oz., is very soft, dark purple in colour. It contains several spheroidal masses the size of large peas, of firmer consistence than the rest of pulp, and of darker colour.

Adrenals normal.

Kidneys. Right 4 oz.; left 6 oz. In both are numerous, small yellowish areas of irregular shape; they are not obviously caseous. Rest of tissue normal.

The prevertebral, abdominal, and pelvic lymphatic glands, those at the upper part of both inguinal regions, and those in the neck are much enlarged. They are of soft consistence, but imbedded in fibrous tissue, so that the prevertebral mass feels firm as a whole. In some a few yellowish areas exist.

Prostate and bladder normal.

Bones normal.

Microscopical examination:

Lung.—Chronic pneumonia, with areas of fibro-caseation. A few giant-cells.

Kidney.—Caseation and cell infiltration areas with a few giant cells.

Glands.—Hyperplasia, interstitial fibrosis, areas of caseation, necrosis. In centre of the necrotic areas of glands are large crowds of bacilli with staining reaction of *B. tuberculosis.* The bacilli are larger than the ordinary human tubercle bacilli; they take the stain very deeply; they are massed in

the central portion of the necrotic focus; on the outer parts they are quite discrete.

In the *lung* there are a few bacilli, but none are found in the kidney (one section).

Case 2.—J. P., æt. 21, hotel porter. Admitted July 21, 1903.

Patient was admitted for pain in the chest. The patient has not been well for twelve months, and his mother has noticed that he has been getting thinner.

On July 19 he complained of pain across the back, and the next day he had more pain on the left side. In the evening the pain increased in severity, and he was unable to get about.

The patient has had some cough and shortness of breath for six months, but he has been able to go on with his work until the day before admission.

There has been no headache, epistaxis, or vomiting.

Five years ago the patient had an attack of acute rheumatism, and was laid up for fourteen days. He has had several slight attacks of rheumatism since. He has had no other serious illness.

Family history.—Father died of phthisis aged fifty-one eighteen months ago. Mother living and healthy. She has had eight children; two died of whooping-cough. Patient used to sleep in the same room as his father when the latter was ill.

The patient is thin and anæmic. He is rather deaf, but this is not of recent origin. Pupils are equal. The cardiac impulse is over the fifth space in the nipple line. The area of cardiac dulness extends up to fourth rib, not to right of sternum. There is a loud mitral systolic murmur conducted into axilla. An aortic diastolic murmur is audible at the base and down sternum to xiphoid. It is most loudly heard over fourth left costal cartilage. Pulse is regular, quick; low tension. There is marked idiomuscular contraction all over both apices. The percussion note is flat at both apices, in front and behind. A few râles can be heard at the end of deep inspiration in the second left space in front, and a crepitant râle in second right interspace in front of chest. His appetite is fair. The tongue is coated and dry down midline. The abdomen is rigid. The liver is three fingers' breadth below costal margin in nipple line. Bowels are

regular. There is no vomiting. No œdema of legs. Urine 1020, acid; free from albumen and sugar.

On August 8 it was noted that the patient had occasionally been delirious, and had vomited twice.

September 14.—Patient says that he is feeling better, but complains of aching pain in his back, which he says is extending down his legs. There is some slight fulness over patient's left buttock, and slight heat as compared with other side, but no fluctuation can be detected.

28th.—The pains have nearly all disappeared except about the chest. He sleeps well, his appetite is good, his tongue is clean, and the bowels are regular.

October 5.—Patient says that he is feeling quite comfortable, and is no longer troubled with pain except slightly in the legs after he has been up and about the ward. Sleeps fairly well.

19th.—Patient feels quite comfortable. He has no pain. The condition of the heart is unchanged.

November 2nd.—Patient is still free from pain, and says that he is feeling all right. Impairment of resonance is noted at the right apex; a few sibilant rhonchi and crackling sounds are heard.

9th.—It was noted that the abdomen was still rigid. No enlarged glands were detected.

16th.—Patient complains of a troublesome, dry cough.

18th.—Sibilant rhonchi and râles all over both lungs, most marked at right apex. There is some dyspnœa.

23rd.—Patient has increasing dyspnœa, and complains that sometimes he can hardly get his breath. He has a very troublesome cough, but without expectoration. The impairment of resonance at the right apex is more marked. The cardiac physical signs are unaltered. Pulse 140; low tension; dicrotic. There is some œdema of the ankles.

The patient gradually grew worse, the dyspnœa increased, and the cough continued troublesome, but without expectoration, and he died on November 27.

Three examinations of the sputum were made on August 7, September 29, and October 21 respectively, but tubercle bacilli were not found.

On September 15 cultures from blood in broth gave a pure growth of an oval coccus, chiefly in pairs, but also in short chains of four. Subcultures on agar gave a delicate film of small translucent colonies. The organism stained by Gram, and was distinctly lanceolate in shape.

From these characters it is probably the *B. pneumoniæ*, the delicacy of the subcultures being in accordance with this view. Staphylococci and streptococci are absent.

On October 9 an examination of the blood was made, with the following result:

Red corpuscles . . .	3,900,000
Leucocytes	6,400
Polymorphonuclears .	62 per cent.
(Eosinophils 1 per cent.)	
Monomorphonuclears .	17 „
(Large 7 per cent.)	
(Small 10 per cent.)	

The urine was repeatedly examined; the specific gravity varies from 1014 to 1022. It never contained sugar, and only a trace of albumen on one occasion.

Throughout nearly the whole of the patient's stay in the hospital the *temperature* was of a hectic type, the evening temperature being 102° to 103°, and the morning temperature being usually between 98·4° and 99°. On nine occasions only did the evening temperature exceed 103°, and the highest temperature recorded was 103·8°.

From August 25 to September 12 there was a distinct improvement in the temperature. The highest temperature during this time was 102°, the usual evening temperature being about 101°. Then followed a period of higher temperatures, rising to 103° and upwards in the evening. From October 13 to 20 there was a period of comparatively low temperature; for three days it did not rise above normal; from the 20th to the 24th it oscillated between 96° and 103°; then came two days of normal temperature; then followed three weeks of hectic temperature, an evening rise to about 102°, with a morning temperature of 97° to 100°. Two days before death the temperature fell to 95·4°, and gradually rose, and on the day of death reached 103°.

Necropsy made fourteen hours after death.

Body emaciated. Slight œdema of feet and legs. Pale lividity of skin. Herpes of upper lip. Yellow discoloration of skin of upper part of thorax.

Much yellow serous fluid in pleural, pericardial, and peritoneal sacs.

Lungs large, subcrepitant, partly from red induration, partly from early pneumonia; the latter portions are dark

red and friable, the former tough. Œdema; muco-purulent fluid in bronchi.

Heart 22 oz., shape globose, in diastole; left ventricle dilated and hypertrophied; right heart hypertrophied. Aortic and mitral valves thickened and covered with coarse vegetations, which have spread along chordæ to wall of ventricle and upwards into auricle. Part of mitral is ulcerated. Much soft blood and decolourised clot in cavities.

Arteries normal.

Liver 6 lbs. 2 oz., nutmeggy, soft.

Pancreas 5 oz., normal. Spleen 18 oz., adherent to diaphragm; large old, and recent small infarcts.

Kidneys: each 8 oz.; numerous red infarcts. "Cardiac," tough, red, mottled with yellow.

Adrenals normal.

Bladder normal. *Testicles* normal.

In an interesting communication of "A Case of Ulcerative Endocarditis," read by the late Dr. W. M. Ord before the Medical Society of London,* he pointed out that the following conditions may give rise to remitting or paroxysmal pyrexia.

1. Ague.
2. Remittent fever.
   (*a*) Malarial.
   (*b*) Non-malarial, chiefly intestinal.
3. Enteric.
4. Relapsing fever.
5. Tuberculosis.
6. Confined pus.
7. Pyæmia.
8. Septicæmia.
9. Ulcerative endocarditis.
10. Lymphadenoma.
11. Occasionally leucocythæmia.
12. Impacted gall-stones and irritation of the biliary passage.
13. Irritation of the urinary passages.
14. Irritation of the alimentary canal.
15. Syphilis.
16. The morphia habit.
17. Foreign bodies impacted in the tissues.

To this list I would add malignant disease and post-influenzal cases.

* *Medical Society's Transactions*, vol. xi, p. 274.

In any given case of prolonged pyrexia, the possibility of its being due to any one of the above enumerated nineteen conditions, should be borne in mind, until by a process of exclusion the diagnosis is narrowed down to two or three at the utmost.

It will be found in practice that in this country these cases of protracted pyrexia are more commonly due to tuberculosis and malignant endocarditis than to any other two conditions. It is therefore to draw attention to the frequency with which these two conditions have been confounded, and the means by which a diagnosis may be arrived at, that I have brought forward the two cases the notes of which I have given.

Neither the age nor the sex of the patient will help us, as the diseases spare neither age nor sex, and their respective age and sexual incidence will furnish no clue. Family history may be of a certain assistance, as the occurrence of several cases of tuberculosis among the immediate relations of the patient should awaken suspicion. On the other hand, there may be a family proclivity to rheumatic affections, and then the probability of a rheumatic endocarditis may be entertained. The past history of the patient will often give valuable information, as, for instance, attacks of hæmoptysis (it must be remembered, however, that free hæmoptysis not unfrequently occurs in cases of mitral stenosis), pleurisy, fistula in ano, enlarged glands, etc., may lead one to suspect tuberculosis. The history of a pneumonia is of equivocal value. Pulmonary tuberculosis may supervene upon lobar pneumonia, especially of the apex of the lung, but over against this tendency may be set the fact that pneumonia is one of the most common antecedent conditions of ulcerative endocarditis. The history of acute rheumatism followed by cardiac trouble should incline the scale strongly in favour of ulcerative endocarditis.

In looking at Case 1, the evidence is strongly in favour of the diagnosis of pulmonary tuberculosis which was originally made, in spite of the history of rheumatism. The patient had had enlarged glands, cough, with expectoration of blood and loss of weight, and there were slight physical signs at the right apex posteriorly. All that was wanted to clinch the diagnosis was the detection of the tubercle bacillus in the sputum. This confirmation, however, was wanting, so other possible causes for the pyrexia were considered. The enlarged glands suggested the possibility of leucocythæmia or lympha-

denoma. The former was excluded by the result of the blood-count, and though there was some enlargement of the supra-clavicular glands, the glands originally affected remained unaltered, so that lymphadenoma was put out of court. There practically only remained malignant endocarditis, and, as if to lead one astray, a soft systolic murmur was detected in the third left interspace. The blood had been previously examined, but no micro-organisms had been found in it. The points which this case impressed upon my mind were—first, the importance of repeated examinations of the sputum, and not being content with one or two negative reports; secondly, that a murmur, not accompanied by other evidences of heart disease, should not receive much attention; and, thirdly, the existence of enlarged glands as an evidence of tuberculosis.

The *post-mortem* examination explained the difficulty of diagnosis, and also accounted for the failure to detect tubercle bacilli in the sputum. It is interesting to note what comparatively small pathological lesions will account for prolonged and persistent pyrexia.

Case 2. In this patient the history of acute rheumatism and the physical signs of aortic and mitral disease bulked largely in favour of malignant endocarditis as the cause of his pyrexia; but, on the other side, it was to be noted that the patient had slept in the same room with his father, who had died eighteen months previously of phthisis, and that the physical signs at both apices were suggestive of commencing pulmonary tuberculosis. In view, however, of the fact that three examinations of the sputum gave a negative result, the idea of tuberculosis was dismissed, especially as cultures from the blood gave a growth of an oval coccus, probably the *diplococcus pneumoniæ*. There is another point in the diagnosis on which I would insist, and that is the comparative rarity of the association of valvular disease with pulmonary tuberculosis. Rokitansky went so far as to say that "Persons labouring under enlargement of the heart, whether primary or superinduced by mechanical obstruction at its orifices, do not contract tuberculosis." Though it is uncommon to find phthisis and heart disease in the same person, the statement of Rokitansky, just quoted, is of too sweeping a character, though there is one form of valvular disease, viz. mitral stenosis, which seems to be an almost complete bar to the development of phthisis. For instance, Percy Kidd gives one instance only of mitral stenosis in 500 necropsies, and Walsham one in 130. Curiously enough, however many cases of

mitral stenosis show signs which can easily be mistaken for pulmonary tuberculosis, and to lead the unwary astray the disease is often accompanied with hæmoptysis. I have had cases sent to me as advanced consumption which were really examples of mitral stenosis with lung signs and symptoms. Hence it follows that the physical signs usually met with in pulmonary tuberculosis, such as dulness on percussion, with râles at the apices of the lungs, should not, in a person suffering from mitral stenosis, be accepted as evidence of pulmonary tuberculosis unless the diagnosis has been made certain by the discovery of the tubercle bacillus in the sputum. Given, therefore, the signs of valvular disease with enlargement of the heart, and especially if mitral stenosis be present, it would be unwise to make a diagnosis of pulmonary tuberculosis unless tubercle bacilli were present in the sputum, and in the absence of these bacilli the explanation of a hectic temperature should be sought rather in a malignant endocarditis than in tuberculosis; and it is by such a train of reasoning that one has to arrive at a diagnosis in those cases in which blood cultures give no definite information.

XXXIII.—*On some Cases of Partial Heart-block.* By A. M. Gossage, M.D. *Read May* 24, 1907.

THE recent work of Tawara (*Das Reizleitungssystem des Saugetierherzens*, Jena, 1906) and Keith (*Lancet*, 1906, ii, p. 359), confirming the view of Stanley Kent (*Journal of Physiology*, 1893, xiv, p. 43) and His (*Arbeiten aus der medicinischen Klinik zu Leipzig*, 1893), has proved the existence of a muscular connection between the auricles and ventricles in mammalia and man and so has removed the last obstacle to the application to the human heart of the conclusions of Gaskell (*Schäfer's Physiology*, vol. ii, article on the heart) and Englemann (*Pflüger's Archiv*, vol. lxv, 1897, p. 535) as to the causation of the cardiac rhythm. The auriculo-ventricular bundle starts in a network of muscle fibres in the auricular septum and passes down to the fibrous portion of the inter-auricular and inter-ventricular septum, whence it descends between the ventricles as a well-defined band, separated from the rest of the cardiac muscle by fibrous tissue, and divides into two branches, one to each ventricle, each of which splits up into a number of smaller branches to be distributed to the musculi pectinati and to the ventricular walls. It is to be noticed that the bundle ends not at the base of the ventricles but at the apex and it is probable that the ventricular contraction starts at the apex and not at the base.

A particular group of symptoms has been observed not very infrequently since their first description by Adams (*Dublin Hospital Reports*, iv, 1827, p. 396) and Stokes (*Dublin Quarterly Journal of Medical Science*, 1846) and has received the name of the Stokes-Adams syndrome. I have myself seen six examples of this syndrome. The symptoms are a very infrequent pulse associated with certain nervous attacks, such as giddiness, syncope, epileptiform seizures, or even attacks like apoplexy. The syndrome is commonest in elderly people with marked arteriosclerosis, but it may occur in young adults, as in one of my own patients, who was only twenty-four. The infrequency of the pulse is usually persistent, but sometimes it only comes on during the nervous attacks, whilst in other cases the infrequent pulse and the nervous attacks, after being present for several months, may

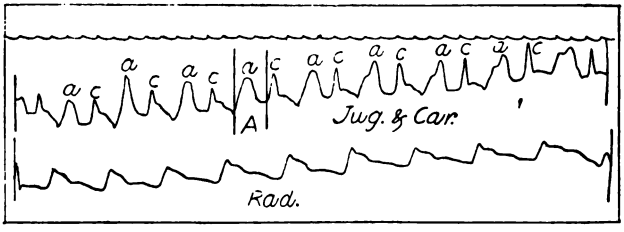
completely disappear. In Case 3 of those reported in my paper in the *Transactions of the Clinical Society,* vol. xxxviii, the symptoms lasted four months and then quite suddenly the pulse-rate became normal and the patient had no further nervous attacks during the next fifteen months, after which time he was lost sight of. Naturally such recoveries are more common in younger patients, whose symptoms have started after some acute disease. During the more severe attacks the ventricles generally cease to beat, sometimes for nearly a minute, and the stoppage of the circulation precedes the cerebral symptoms and is probably the cause of them.

Stokes noticed that a marked pulsation of the veins of the neck developed in his patient and the same phenomenon has been reported by a number of other observers, several of whom noticed that the venous pulsations were more numerous than the arterial, and it was even suggested that the auricles were beating faster than the ventricles (Chauveau, *Rev. de Med.*, Paris, 1885, v, p. 161). His in 1899 (*Deutsch. Archiv für klin. Med.*, vol. lxiv, 1899, p. 329) came to the conclusion that the essential lesion was a depression or abolition of conductivity in the fibres joining the auricles and ventricles, so that the ventricles only beat once to every two, three, or more auricular beats. This explanation is in striking agreement with the experimental results of Gaskell, who by compressing the connecting strand of muscle between two larger pieces and then stimulating one of the latter was able to "block" the stimulus, so that the other piece only responded to every second, third, or fourth stimulus, or did not respond at all, there being, as Gaskell said, either partial or complete "heart block" (Gaskell, loc. cit.). Later researches have completely demonstrated the correctness of His's hypothesis and have shown that there may be partial or complete "heart block" in human beings where the ventricles only respond to every second or third auricular beat, or where the auricles and ventricles beat quite independently of one another. Pathologically several cases have now been reported which during life showed this syndrome and in which after death disease implicating the auriculo-ventricular band has been found. This view has been further confirmed by Erlanger's experiments on dogs in which he produced heart block by compressing the auriculo-ventricular band. More recently Erlanger (*Johns Hopkins Hospital Bulletin,* July, 1906) has damaged this bundle in a living dog and managed

to keep it alive for some weeks; the dog exhibited symptoms which closely resembled the Stokes-Adams syndrome; its auricles beat at about 140 per minute and the ventricles at only 60, while occasionally the ventricles ceased beating for an appreciable period.

One can demonstrate that the auricles are beating faster than the ventricles in several ways. Wenckebach was able to prove the dropping of single ventricular beats by carefully timing the individual intervals. Nowadays we can see the heart beating by means of the X rays and it is possible to count two, three, or four auricular beats to each ventricular one (it is usually necessary to cover the ventricle in order to see the auricles beating, but these can be compared to the pulse at the wrist). Gibson (*Brit. Med. Journ.*, 1906, vol. ii, p. 22) was able to show that the auricles were beating two to

Fig. 1.

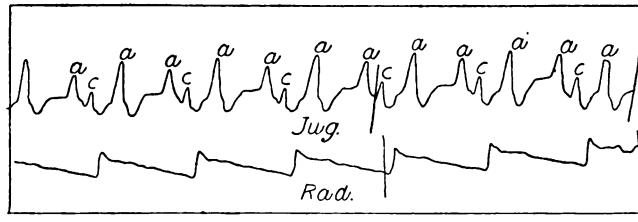


Depression of conductivity by digitalis. (*After Mackenzie.*)

three times as fast as the ventricles in one of his cases by observing the electro-motive changes of the heart by means of the capillary electrometer. The most convincing method, however, is that of Mackenzie, who records at the same time the pulsations of the jugular vein and of the radial pulse. In the jugular pulse, when obtainable, the auricular beat is shown preceding the wave due to the carotid and the condition of the conductivity of the cardiac muscle can be estimated by the interval between these two waves (see Fig. 1). If conductivity is depressed the stimulus takes longer to pass from auricle to ventricle and this interval is lengthened. In Fig. 1, where *a* is the wave due to the auricular beat and *c* the wave due to the carotid, the interval between *a* and *c* instead of the normal one-fifth of a second is shown by the time-marker to be two-fifths of a second and therefore conductivity is depressed. On giving digitalis to this case Mackenzie,

who has kindly allowed me to use his tracings, found that the pulse was halved in frequency and the sphygmographic record shows that only every other auricular beat is followed

FIG. 2.



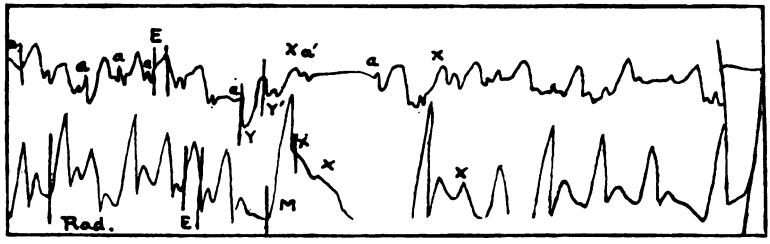
Depression of conductivity by digitalis. (*After Mackenzie.*)

by a beat of the ventricles (see Fig. 2), the further depression of conductivity produced by the digitalis causing a block after every other beat.

In animals it is well known that digitalis depresses conductivity and here Mackenzie has afforded us with a striking proof that the same holds good for man (*Brit. Med. Journ.*, 1905, vol. i, p. 519). Clinically, we find that conductivity may also be depressed by certain acute illnesses, *e. g.* rheumatism and influenza, and the whole of the Stokes-Adams syndrome may follow such a febrile attack. In animals stimulation of the vagus depresses conductivity and Mackenzie has recently shown (*Brit. Med. Journ.*, 1906, vol. ii, p. 1110) that this may occur also in man; in a patient whose conductivity was shown to be depressed by the length of the *a*—*c* interval, swallowing induced a further depression so that certain stimuli from the auricles were blocked and some of the ventricular beats dropped: the stimuli from the pharynx and œsophagus must have affected the heart reflexly through the vagus. After a prolonged rest the conductivity improves, as is shown by the shortening of the *a*—*c* interval; so that after an intermission conductivity may be sufficiently recovered to let the next stimulus through. Frequent stimulation would exhaust all the properties of the cardiac muscle, so that ventricular intermission would be more likely to occur in cases of depressed conductivity with frequent heart rhythm than with infrequent. In Fig. 3 an intermission is shown taking place after two extrasystoles, both of which occur very early. Conductivity was probably depressed in this case, though in the absence of a venous pulse positive demonstration is

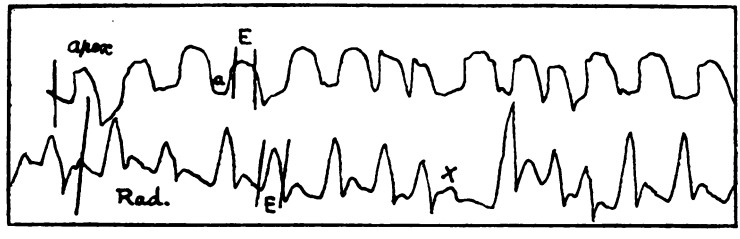
lacking. In the tracing the radial pulse and a cardiogram were taken at the same time, but the receiver was not placed over the exact apex of the heart, but somewhat more internally and the commencement of the ventricular systole is marked by a fall instead of a rise and with a strong systole,

Fig. 3.



as at *y*, the fall is very marked. The ventricular systole is preceded in the cardiogram by a wave *a*, which is probably due to the auricular systole, though it is much more marked than the auricular wave usually is in a cardiogram, and is much more marked than in any other tracings taken from the same patient (see Fig. 4 where the cardiogram was taken from

Fig. 4.



the apex on the same day and is of the normal type). The heart action in this case was very irregular due to numerous extrasystoles. At *M*, in Fig. 3, there is a large contraction after a compensatory pause following three extrasystoles and this contraction is succeeded by two very early and therefore small extrasystoles, *x* and *x* in radial record and *y'* and *x* in cardiogram. In the cardiogram the second of these is followed by a small wave, similar to the waves *a* described above, and then there is a pause showing the absence of at least one ventricular contraction.

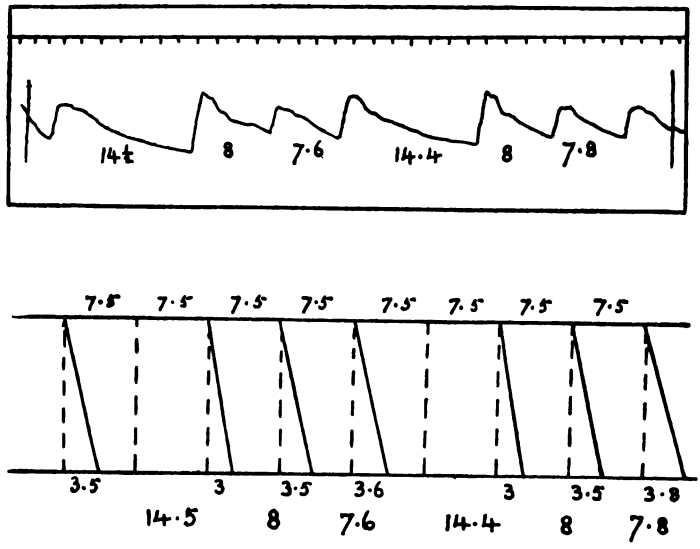
CASE 1.—L. N., a girl, æt. 16, has been under my care for about six years suffering from stenosis and incompetence of the mitral valve and adherent pericardium. Her heart is much dilated and hypertrophied; the apex-beat, heaving and diffuse, is in the fifth intercostal space, an inch and a half external to the nipple, while the absolute cardiac dulness extends to the upper border of the fourth rib and to an inch and a half to the right of the right sternal border; there is also marked præcordial projection. Lately the area of cardiac response has become more limited and the cardiac rhythm has become very irregular, as is shown in Figs. 3 and 4, but no intermission has been noticed except that recorded in Fig. 3.

It is to be noted that all cases of depressed conductivity do not necessarily exhibit a block at the auriculo-ventricular junction, this only occurring in the more severe cases, while the Stokes-Adams syndrome only occurs in the more severe cases of heart block.

In Fig. 5 an example of slight depression of conductivity is exhibited in which the Stokes-Adams syndrome was absent. The tracing shows after every second normal beat an apparent intermission, which may be either due to a very early extrasystole, too small to cause a wave in the radial pulse, or to the dropping of a ventricular beat, a true intermission. In the dying hearts of frogs Engelmann (*Pflüger's Archiv*, Bd. 52, 1894) has shown that conductivity is depressed and that occasionally beats of the ventricle may be left out: in these experiments the prolonged interval produced by ventricular intermission was always less than double of the normal interval and the prolonged interval was followed by a beat which was longer in duration than a normal beat, while the interval between auricular contraction and ventricular gradually increased with successive normal beats. Wenckebach has described intermissions in human hearts of precisely the same character (*Die Arhythmie*, p. 67) and has attributed them to depression of the property of conductivity. In cases of what used to be called intermittent action of the heart, but which are really due to early extrasystoles, the radial tracing is quite different in character. This is well shown in Fig. 6, from a patient, aged 53, with arterio-sclerosis, where an extrasystole occurs after every second normal beat: the other beats here are shown by the time marker (which here and in the other tracings shows intervals of one fifth of a second) to be all of

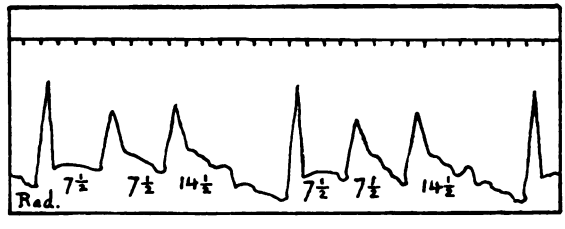
them of the duration of 7·5 tenths of a second. In Fig. 5, however, these normal beats vary in length, and the explanation of this is given in the diagram below the tracing. In

Fig. 5.



this diagram the auricular beats are represented by dotted lines and the ventricular beats by continuous lines between the two horizontals, while the interval between the auricular

Fig. 6.



beats and the ventricular beats is given below on the lower horizontal. The interval between two successive auricular beats is taken as 7·5 tenths of a second and the ventricular intervals are shown by the time marker to be in tenths of a second, 14·5, 8, 7·6, 14·4, 8, 7·8. Conductivity being de-

pressed, the auriculo-ventricular interval would be a long one, 3·5 tenths, and following this the second ventricular beat is dropped out; the long rest afforded the conductivity results in its recovery, so that the next *A.*—*V.* interval is much shorter, three tenths; the third contraction results in a marked depreciation of the conductivity, so that the third *A.*—*V.* interval rises to 3·5 tenths of a second; the effect of the fourth contraction, just as in Engelmann's experiments, is not so marked, the *A.*—*V.* merely rising to 3·6 tenths; this is followed by another ventricular intermission, and consequent recovery of conductivity. This case, therefore, conforms both with the phenomena found with depressed conductivity in experiments and with Wenckebach's clinical cases, though the alterations in the conductivity, depressed on the one hand by successive contractions, or benefited on the other by prolonged rests, is less striking than in his patients.

Case 2.—E. C., æt. 36, charwoman, came to the hospital complaining that for a week she had been suffering from shortness of breath, inability to lie down in bed and pain under the left breast. She also had some pain in the left knee and her feet were a little swollen. Three years ago she had a similar attack, from which she had rapidly recovered, but she had never had rheumatism or any other serious illness. For the last two months she had had amenorrhœa; she had never been pregnant.

On examination she was a well-nourished woman, with a rather flushed face. The apex of the heart was normal in position and the cardiac dulness was normal. The heart sounds were weak and at the bottom of the sternum there was a rough scraping noise with the systole, evidently pericardial in origin. The heart intermitted at times, no intermediate small sound being heard. The pulse tension seemed somewhat raised. The urine had a specific gravity of 1008, an acid reaction and contained a trace of albumen (see Fig. 5).

If the property of conductivity be still further depressed every other beat of the ventricle would be dropped out instead of every third beat, or the ventricle might only respond once to three auricular beats. With complete abolition of conductivity in the *A.*—*V.* fibres, auricles and ventricles would beat independently of one another; several clinical cases of this

kind have been described. In Fig. 7 an example is given of a case in which every other ventricular beat is dropped. That the auricles were, in this patient, beating twice as fast as the ventricles was proved in several ways. When the case was first seen the rate of the pulse was sometimes 68 and at

FIG. 7.

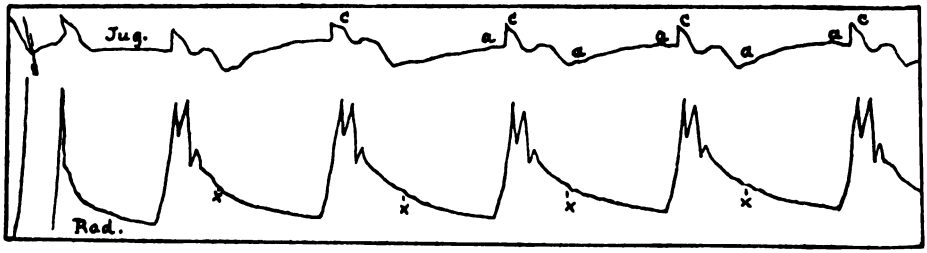


FIG. 8.

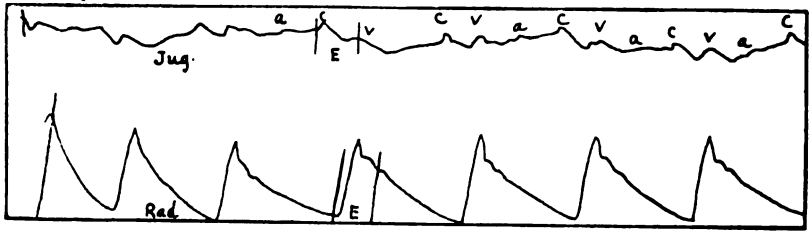
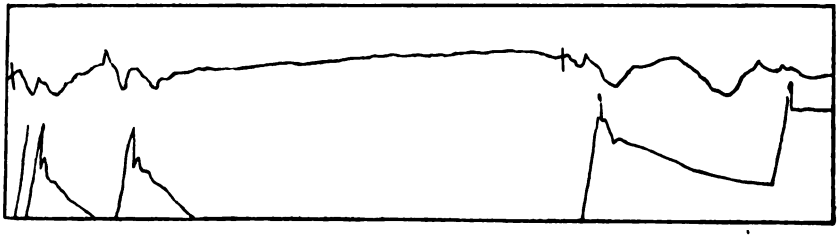


FIG. 9.



other times 34 and the transition from one rate to the other took place quite suddenly (see Figs. 8 and 9). In Fig. 8, where the pulse-rate was 68, a tracing had been obtained from the jugular, which shows, unfortunately not very definitely, an auricular wave *a* before each carotid wave *c*. The exact timing of these waves is ascertained from the radial pulse; the sphygmic period *E* in the latter corresponds to the

period *E* in the jugular pulse and at the commencement of the sphygmic period in the jugular tracing is the wave *c* due to the carotid and preceding the sphygmic period is the auricular wave *a*. The interval between *a* and *c* is very much longer than the normal, showing a grave depression of conductivity. Fig. 7 also shows in the tracing from the neck a wave *a* before the carotid wave *c* and this is probably due to the auricle. The *a*—*c* interval here is normal in length, as the infrequent rate allows ample time for the conductivity to recover after each contraction. There is another wave *a* between the adjoining carotid waves and this corresponds to the position that the auricular wave would be expected to occupy if the auricles were beating regularly at twice the rate of the ventricles. These auricular waves are, however, very indefinite, and much stress cannot be laid on them. In the radial tracing a depression *x* is to be noticed sometimes: Mackenzie has demonstrated that a depression of this kind is connected with the beat of the auricles; probably the auricular beat sends a wave through the quiescent ventricle to the aortic valves and to the blood in the aorta, which is followed by a rebound giving rise to the depression. In the present case the most striking proof, however, was afforded by seeing the auricles, by means of the X-rays, beating at double the rate of the pulse.

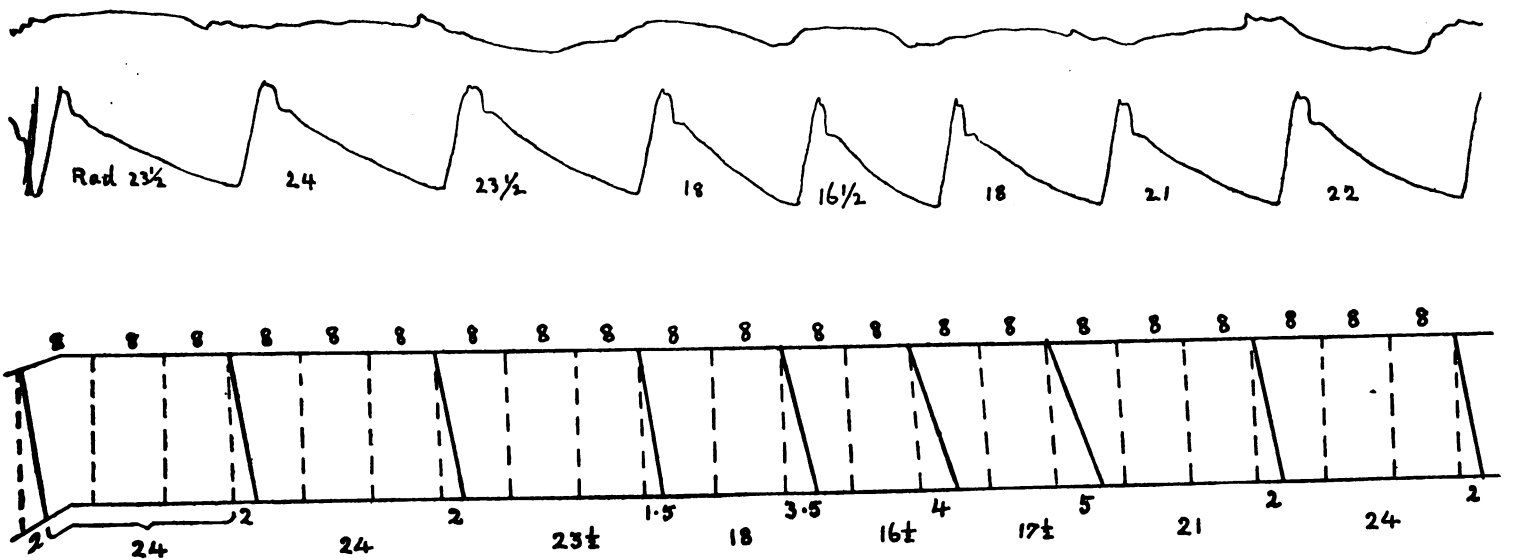
Case 3.—J. B., a clerk, æt. 58, was shown to the Clinical Society on October 26th 1906 in conjunction with Dr. Percy Northcote. He was first seen in January 1906 and has been examined frequently since; he has also been under continuous observation as a patient in the hospital on two or three occasions. At the age of twenty-one he had had syphilis, but had otherwise been healthy until 1902 when he had a sudden attack of unconsciousness, falling and injuring his head. Since then he has had numerous similar attacks, more frequent at times when he is overworked or worried. The attacks are either a momentary giddiness, with or without falling, or complete unconsciousness for several seconds, during which time his head is said to roll and his limbs to twitch. He had been aware for about four months before he came under observation that his pulse was beating at the rate of about 30 per minute. Throughout his illness he has felt the cold very severely.

On examination the arteries were found to be much thickened and the tension decidedly increased, the sphygmo-

manometer showing that the systolic blood-pressure was equivalent to 178 mm. of mercury. The urine generally contained a small quantity of albumen. Nothing abnormal was found in the heart except the rhythm. When first seen the ventricles were beating at 68 per minute, but the patient suddenly felt giddy and the rate dropped to 32—*i. e.* to about half the former rate. This sudden halving of the pulse-rate was repeated on several occasions while he was under examination, the change being generally, but not invariably, associated with giddiness. As a rule the pulse-rate was between 26 and 36, although it occasionally rose to over 60, but for twelve months now it has never been found to exceed 36. On several occasions the patient was kept for some days in bed under observation and on every occasion he was benefited by the rest. No severe attacks took place while he was under observation, but there were numerous slighter ones, from momentary giddiness to attacks in which he would have fallen if not supported, and it was noticed that these latter seizures were associated with a cessation of the ventricular beat for at least five seconds (see Fig. 9). On no occasion were any intermediate sounds heard between the larger ventricular beats.

An example of a condition where the conductivity is still further depressed is afforded by the patient from whom Fig. 10 was taken. In this tracing the pulse is as infrequent as in Fig. 7, but it differs from the latter in being very irregular. Unfortunately we were not able to obtain a tracing with the time marker, so that the conclusions as to the length of the individual intervals is rather hypothetical, but the movement of the paper in the sphygmograph was probably not so irregular as to impair their general correctness. It is assumed that the auricles are beating at the rate of 75 per minute and that, therefore, the duration of an individual auricular beat is eight tenths of a second and a diagram of the tracing similar to that in Fig. 5 has been constructed on this supposition. The dotted lines represent auricular beats and the continuous lines beats of the ventricles and the intervals between auricles and ventricles are given on the lower horizontal. The first interval between two successive beats of the ventricles is a long one, twenty-four tenths: probably two ventricular beats are here missing and ample opportunity would be afforded for the conductivity to recover, so that the *A.*—*V.* interval would be short, two tenths: this

FIG. 10.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2

also applies to the second interventricular interval; the third interval also shows the dropping of two beats of the ventricles, but is shorter than the previous ones, because the conductivity, after so many long intervals, has so far recovered that the *A.*—*V.* interval is only 1·5 tenths (compare Engelmann's experiments on the dying heart of the frog). The conductivity having so far recovered, on the next occasion only one ventricular beat is dropped out and, in consequence of the shortened interval, there is a marked increase in the *A.*—*V.* interval, which rises to 3·5; another interval, with the one beat of the ventricles dropped, ensues and, as in Engelmann's experiments, is associated with a further depression of conductivity, though only a slight addition to the *A.*—*V.* interval occurs. Again one ventricular beat is dropped out and the *A.*—*V.* interval rises to five tenths and after this grave depression of conductivity on the next occasion two ventricular beats are dropped, with a consequent recovery of conductivity and a lessening of the *A.*—*V.* interval to two tenths again. The auricles beating regularly at intervals of eight tenths the inter-ventricular intervals should be successively 24, 24, 23·5, 18, 16·5, 17·5, 21, and 24. The actual intervals marked on the tracing are 23·5, 24, 23·5, 18, 16·5, 18, 21, and 22, and the slight differences between the actual and expected are probably due to instrumental errors.

Case 4.—Mr. K., æt. 47, was kindly shown to me by Dr. Archibald Garrod, who adds to his kindness by giving me permission to record the case. In 1896 the patient had an attack of syphilis, but otherwise had enjoyed good health up to three years ago, when he was suddenly attacked with faintness while riding and this attack was followed by a succession of others so that it was with great difficulty that he reached home. He put himself under the care of a doctor who shortly afterwards told him that his pulse was very infrequent. Since this sudden onset he has been liable to similar attacks and at times these have been very frequent. He has, however, after having been treated with large doses of iodide of potassium and in the enjoyment of a holiday, had no attacks for the last two months, but his pulse, as always, has remained very infrequent. The seizures come on suddenly and the patient falls, loses consciousness momentarily, but, so far as he knows, does not struggle or twitch. When first seen by Dr. Garrod his pulse-rate was 27 and the systolic blood-pressure was equivalent to 130 mm. of mercury and

there was a trace of albumen in the urine. The apex-beat of the heart was internal to the nipple and not particularly forcible; a soft systolic murmur was heard all over the heart, loudest at the base and conducted into the great vessels of the neck. No intermediate sounds between the obvious forcible beats were heard and there was no visible pulsation of the veins of the neck. At the time the tracing (Fig. 10) was taken the pulse was 30 and no tracing was obtainable from the veins of the neck.

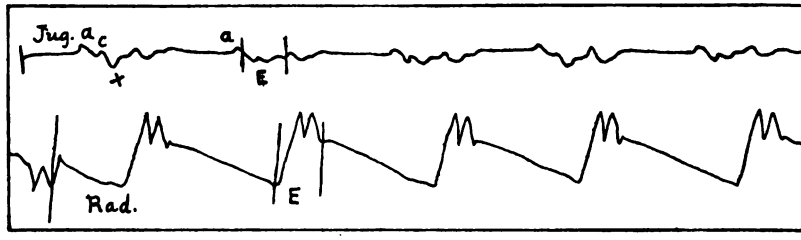
The nervous attacks of the Stokes-Adams syndrome are associated with a further depression of conductivity, so that a succession of auricular beats do not pass on to the ventricles. This further depression of conductivity is possibly due to the influence of the vagus, the vagus being excited by reflex irritation, emotion, etc. In some cases many seconds pass (up to 40) without a ventricular beat and death occurs not infrequently in these attacks (one of my patients died in an attack). An example of a mild attack is shown in Figs. 8 and 9, which were taken from the same patient as Fig. 7. In Fig. 8 the heart is beating quite regularly at 68; while Fig. 9 was being recorded, immediately after the taking of Fig. 8, the patient suddenly felt faint and had to be supported and it is seen that no ventricular beat occurred for five seconds; after the attack the ventricles are shown to be beating at half their former rate. Tracings during much more severe attacks were taken by Dr. Hay from a patient under the care of Sir James Barr (*Brit. Med. Journ.*, 1906, ii, 1122).

In some cases feeble sounds have been heard between the obvious ventricular beats, but this has not been the case in any of the six patients I have seen personally. These sounds have been attributed to the contractions of the auricles. About eighteen months ago I brought forward arguments against the view that these sounds are auricular in origin and I still think that there are many difficulties in the way of accepting it. A more extended study of cardiac phenomena in the light of the myogenic theory has, however, convinced me that the alternative explanation that they are due to ventricular extrasystoles has still greater difficulties in its way.

Bradycardia is not invariably caused by want of conduction of the stimulus from auricle to ventricle. The cardiac rhythm may be infrequent from a slow building up of the stimulus as is not uncommon after acute febrile disorders. Quite healthy

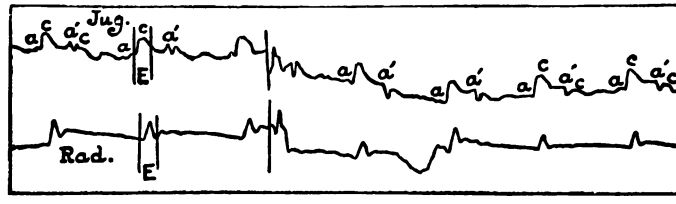
people may normally exhibit a very infrequent pulse which can be recognised as due to this cause if a venous pulse be obtained. Fig. 11 is a tracing from a healthy man whose

FIG. 11.



pulse was usually 40 to the minute and in the jugular part of the tracing it is evident that the auricles are beating just as infrequently as the ventricles. In such cases the phenomena of the Stokes-Adams syndrome are not present. The regular occurrence of an early extrasystole after each normal beat may also simulate a bradycardia. On listening to the heart the extrasystole can often be heard even if no wave be shown in the radial and frequent examinations of the heart will generally show that it is over-excitable. Thus, in Fig. 12, an early extrasystole occurs after each normal beat and is so small that no wave is shown in the radial pulse giving the appearance of a bradycardia, while the venous pulse above shows that the auricles are beating at an ordinary rate; a

FIG. 12.



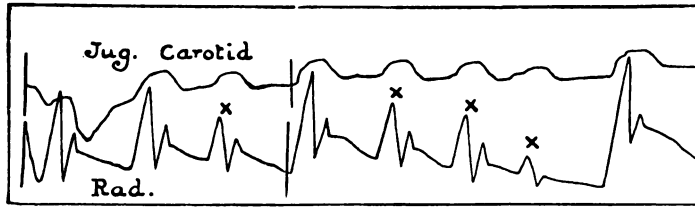
later tracing (Fig. 13) shows numerous extrasystoles and gives the clue for the explanation of Fig. 12.

Depression of the excitability of the ventricles may also lead to their only responding to every other auricular beat, as in the case reported by Hay (*Lancet*, 1906, i, 139). In experiments with frogs it has been found that depression of

contractility leads first to an alternating action of the heart and that later the smaller beats may disappear from the ventricles, so that they only beat at half the rate of the auricles (Straub, "On Antiarin," *Archiv für experiment. Path. und Pharmokol.*, 1901, xlv, p. 347). The digitalis group of drugs also depress contractility in the human heart and may induce an alternating action, but so far the further condition of every other ventricular beat being abolished has not been observed (cf. Hay, however, *Liverpool Medico-Chirurgical Journal*, 1906, p. 49).

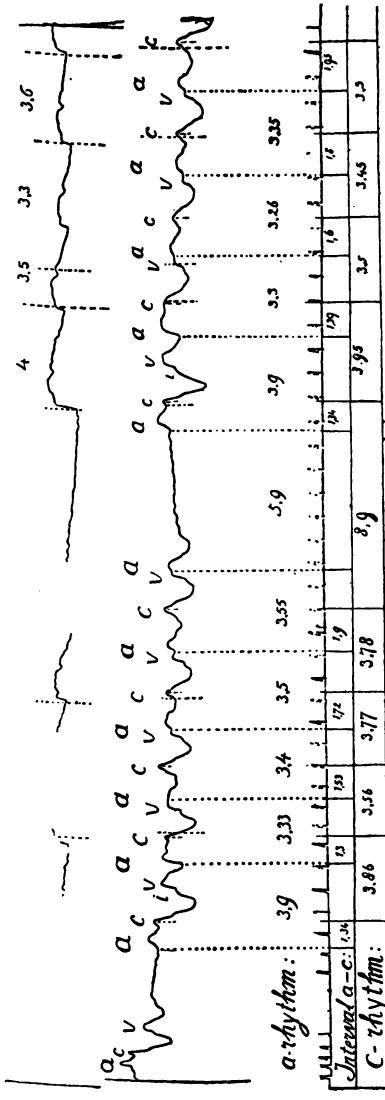
The usual place for a block of the stimulus to occur from depression of conductivity is in the fibres connecting auricles and ventricles and definite disease has been described in

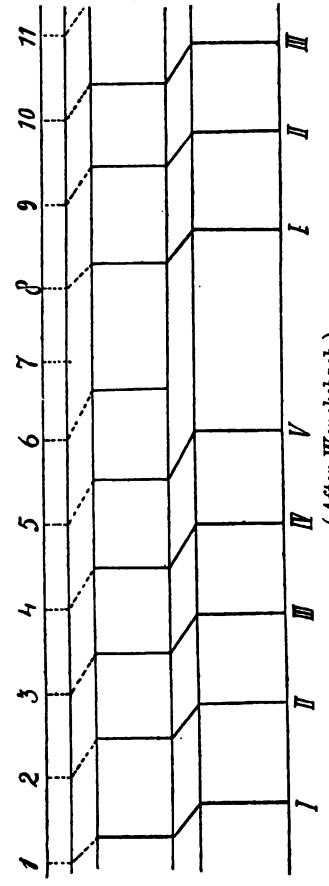
FIG. 13.



this situation in several cases. Recently, however, Wenckebach has given reasons for thinking that there may be depression of conductivity between the veins and the auricles. Professor Wenckebach has kindly allowed me to make use of the diagram shown in Fig. 14, which explains how this conclusion has been arrived at. The diagram shows at the top a tracing from the pulse and below this one from the jugular vein and a time marker in fifths of a second. In the jugular record *a* is the wave due to the systole of the right auricle and *c* the wave due to the carotid pulse. The tracing shows a gradual lengthening of the *a*—*c* interval due to depressed conductivity and then a pause of such length that two beats of the ventricles must be missing; after this interval the conductivity is restored and the *a*—*c* interval is short, but then gradually increases again. After the fifth systole the next auricular beat appears in its proper position, the auricles beating regularly, but there is no corresponding ventricular beat, *i. e.* there is a block at *A.*—*V.* junction just as in Fig. 5. This sixth auricular systole is followed by a prolonged auricular interval in which an auricular systole is missing, but

Fig. 14.





where the normal stimulus has probably started from the veins and been blocked at the junction between veins and auricles. It is difficult to explain the successive dropping out of two beats by a block at the *A.*—*V.* fibres, since, in a case where the conductivity is so slightly affected that most of the beats get through to the ventricles, the conductivity ought to have sufficiently recovered after the first intermission to have let the next stimulus through, whilst a block between vein and auricle gives a sufficient explanation of the second intermission. Below a diagrammatic representation of the explanation is given where *Ve.* represents the venous contraction, *A.* the auricular, and *V.* the ventricular. It is possible that a block at junction of veins and auricles is the explanation of the intermission in Fig. 3. A description of this tracing has already been given, and the difficulties of arriving at any certain conclusions about it emphasised. The interval, following the two extrasystoles *x* and *x* after the beat *M*, is longer than would be expected if only one ventricular beat be missing, whereas it exactly corresponds to what would be expected if there were four extrasystoles, arising at the veins, following *M*, of which the first two are *x* and *x* in the radial record, while the third is blocked at the auriculo-ventricular junction, its auricular beat being possibly shown in the apex tracing at *a'*, and the fourth blocked at junction of veins and auricles. This explanation is, of course, very hypothetical and is only suggested as being a possible one. It is of no assistance in settling the question as to whether a block can take place at the junction of the vein and auricle.

XXXIV.—*Foreign Bodies in the Bronchi.* By F. W. Price, M.B. *Read May* 24, 1907.

At the annual meeting of the British Dental Association at Edinburgh, August, 1895, Dr. F. W. Hewitt read an important paper, entitled "An Inquiry concerning the Safety and Sphere of Applicability of Chloroform in Dental Surgery." The writer had collected the records of all the deaths which had occurred during fifteen years in Great Britain in connection with the use of anæsthetics for teeth extraction. Some valuable and indisputable facts were elicited by the inquiry. Thus, of the thirty-seven fatalities thirty-one were caused by the administration either of chloroform or of some combination containing chloroform; whereas only one was in association with ether, and this could be excluded on the ground that death was undoubtedly the result of mechanical asphyxia chiefly due to malignant disease of the mouth; and only five were in connection with nitrous oxide, and one of these arose from the aspiration of a tooth into the larynx.

In twelve of these nineteen cases some details of the posture of the patient during the administration of chloroform were recorded, and from these it appeared that the patient was either sitting or semi-recumbent in seven of the twelve. From these and other facts Dr. Hewitt draws the conclusion that in respect to risk to life chloroform cannot be regarded as a safe anæsthetic in dental surgery; and that this danger is in large measure due to the fact that the most convenient postures for the extraction of either upper or lower teeth are the most dangerous as far as the administration of the anæsthetic is concerned. But besides this risk to life, it is unfortunately true that the semi-recumbent posture predisposes to the gravitation of teeth towards the back of the throat; while extension of the head upon the trunk, which is such a convenient position for removing teeth from the lower jaw, has the effect of drawing the base of the tongue and epiglottis away from the pharynx and larynx respectively, and consequently of impeding the act of swallowing and rendering the laryngeal aperture more exposed to the entrance of foreign bodies. These dangers are much greater with chloroform than with ether or nitrous oxide. For these

reasons Dr. Hewitt strongly condemned the use of chloroform as an anæsthetic in the vast majority of cases of teeth extraction, and laid down certain specific injunctions which should be adhered to if its administration should on rare occasions be considered absolutely necessary by some operators. Notwithstanding this note of warning, there is reason to believe that the seriousness of the subject is not fully realised, and that chloroform is still much too largely and without proper precautions employed in dental surgery. The case I am about to relate is a sad example of one of the grave results which may follow such a method of procedure. It is that of a young woman to whom chloroform was administered for the purpose of teeth extraction by a medical practitioner in the country. A root of a tooth entered and became impacted in a bronchial tube, and its presence there ultimately caused bronchiectasis. The patient was under hospital treatment for seven months, during which time practically all the known methods of treatment short of operative interference were tried, and yet the course of the disease was a slowly progressive one in a downward direction. There can be no doubt that a fatal termination will be reached within a comparatively few years. Such a case should cause the most careless to consider well the risks of chloroform as an anæsthetic in dental surgery when adequate precautions are not taken.

A woman, æt. 22, was admitted to Westminster Hospital under the care of Dr. Murrell, to whom I am much indebted for permission to record this case, on November 21, 1905. She complained of cough and pain in the chest. There was nothing of note in the family history. The following personal history was obtained:

Patient has always had a poor appetite, but the only other illnesses besides the present are measles during childhood and influenza early in the winter of 1904. She suffered from caries of the teeth, and in September, 1903, chloroform was administered, and twenty-three teeth or stumps of teeth were removed. After the operation patient stayed in bed for a week on account of headache, nausea, and a swollen face. There was no cough at this time. On getting up at the end of a week a hard, dry, and painless cough developed, which gradually became worse, and in November was noticed to be of a paroxysmal character. In February of 1904 patient began to have a sharp pain in the left axillary region and over the left base, sometimes radiating over practically

the whole chest, worse on coughing, on deep breathing, and on lying on the right side. It became so severe that she was obliged to go to bed, the least movement causing pain, and this lasted for about two months. During this time there was pyrexia, the temperature for a few days being about 105°; the cough changed its character, becoming loose and accompanied by frothy, yellowish expectoration, and each day a small quantity of blood was mixed with the sputa. A herpetic eruption also appeared on the left side of the chest. A few weeks later it was first noticed that the expectoration was offensive. On one evening patient had a very severe pain in the chest, and the following morning, about an hour after waking, she was seized with an intense paroxysm of coughing accompanied by a sensation of choking, which persisted for about half an hour, when with a violent effort a piece of a tooth was brought up, after which patient was relieved. This fragment of tooth was later identified by Mr. Glassington as the palatine root of the first upper molar. A feeling of "soreness" in the left side was felt for some time, the cough in general remained the same, and it was noticed to be worse on turning over from the left to the right side. It continued till admission, as also did the pain in the chest.

On admission the patient presented the following condition:—She complains of a pain in the left side of the chest, chiefly in the left axillary region, not constant, being sometimes absent for days at a time, of a sharp character, and said to be worse on coughing and deep breathing. She has a moderately severe cough, which is considerably more frequent when lying on the right side. Sometimes there is an absence of cough for several hours, and then a considerable quantity of sputum is brought up with ease, the expectoration is muco-purulent, distinctly offensive, and after standing for twenty-four hours shows an upper frothy, a middle watery, and a bottom opaque layer, the amount collected during twenty-four hours being $4\frac{3}{4}$ oz. Microscopic examination fails to reveal the presence of tubercle bacilli, elastic fibres, or Charcot-Leyden's crystals. The odour of the breath is slightly offensive, and there is distinct clubbing of the fingers.

The appetite is poor, the tongue is clean, the molars of the right lower jaw are decayed, the other teeth being artificial, and the bowels are constipated. There is an absence of night-sweating. The heart is evidently displaced somewhat

to the left, as evidenced by the position of the right border on percussion and of the apex-beat, which is normal in force, and in the fifth interspace a quarter of an inch external to the mid-clavicular plane. A systolic (hæmic) bruit is audible in the pulmonary area. The general form of the chest is good. On examination anteriorly there is diminution of expansion on the left side, and at the apex the inspiratory murmur is harsh, but the expiratory part is not prolonged. The vocal fremitus is slightly increased in the left axilla. There is diminution of expansion of the left base, and the vocal fremitus is more distinct. Below the angle of the scapula the note becomes impaired, and at the extreme base there is marked dulness. In the eighth space the inspiratory murmur is diminished, in the ninth the expiration is rather feeble and prolonged, and in the ninth and tenth the vocal resonance is considerably increased. Exploratory puncture of the pleural sac fails to draw off any fluid. The physical signs on the right side are practically normal. Radiographic examination of the chest reveals nothing. The temperature occasionally rises in the evening to 99°. The urine is normal.

A diagnosis of bronchiectasis was made. The patient stayed in hospital for about seven months, during which time, besides other forms of treatment, the following were given a good trial—intra-tracheal injections of guaiacol, menthol, and olive oil; intra-tracheal injections of izal and glycerine; a thorough course of creasote, and later of formic aldehyde vapour baths; inhalations of creasote and of carbolic acid by the ordinary method; formic aldehyde in the form of a mixture; and creasote, picis liquida, and sulphate of atropine, each in the form of a pill, the last named being given with the object of diminishing the amount of bronchial secretion. Nothwithstanding the variety of treatment employed, the general course of the disease was a slowly progressive one in a downward direction. There were attacks of pyrexia; the clubbing of the fingers and the fœtor of the breath increased; the expectoration became more purulent and larger in amount; the apex-beat of the heart was drawn half an inch further to the left; there was a distinct advance in the area of abnormal physical signs on the left side, râles were generally audible at the base, and the percussion note of the right side became higher in pitch. The expectoration was examined for tubercle bacilli on many occasions, and the left pleural sac was several times explored, always with a negative result. It

was not thought advisable to proceed to operative measures. Mr. Rickman Godlee, in a lecture based upon his unique experience in this respect at the Brompton Hospital, stated that multiple bronchiectatic cavities are not good cases for present surgical methods, and this case, judged by the extent of the physical signs, clearly came under that category.

There could be no doubt in regard to diagnosis. The history and whole clinical picture clearly pointed to bronchiectasis, mainly of the left lower lobe, the result of the impaction and retention of a root of a tooth in one of the main branches of the left bronchus. Kingston Fowler has rendered great service in regard to the differential diagnosis of morbid conditions of the lungs and pleuræ by insisting on the supreme importance of the position of the apex-beat of the heart. In this case its displacement to the left, in the absence of air and fluid in the right pleural sac and of any cardiac affection, was manifestly indicative of fibrosis of the left lung or pleural adhesions, both of which conditions are so frequently met with in connection with bronchiectasis.

In reading through the records of a large number of cases of foreign bodies in the bronchi, certain facts become apparent.

In the first place, while it is undoubtedly true that as a rule there are prominent symptoms immediately after the entrance of a foreign body into a bronchial tube, the most characteristic of which are severe dyspnœa, a sensation of suffocation, violent cough and cyanosis, yet in by no means a small proportion of cases there is a complete absence of such symptoms, especially if the foreign body be small, smooth, and rounded. For this reason, when a case of unilateral bronchiectasis presents itself in which other well known causes can be excluded, and indeed in all peculiar respiratory disorders in which the cause is obscure, our suspicions should always be aroused, and an exhaustive enquiry be made regarding the history. In the case I have just described, from the time the patient came out of the anæsthetic state till she got out of bed a week later, there were no respiratory symptoms whatsoever. It is probable that the development of a cough was due to the adoption of the upright posture affecting the position of the foreign body. Suspicion should certainly have been aroused when the cough became paroxysmal, and the patient complained of a severe pain in the left side, which was worse on turning on to the right side. Had a sufficiently early diagnosis been made, there is at least a

strong possibility that timely operative treatment would have been successful.

Secondly, it would appear that the nature of the secondary results of the impaction of a foreign body in a bronchus in considerable measure depends on the character of the foreign body. If it be hard, smooth, and clean, as, for example, the peg of a peg-top, the lesions are more likely to be of a chronic kind, such as bronchiectasis and fibrosis of the lung; whereas if the foreign body have the opposite character, such acute morbid conditions as pneumonia, abscess and gangrene of the lung are much more likely to result. Indeed, apart from physical asphyxia, the last three are the most frequent causes of an early fatal termination. The time which is required to elapse before the bronchiectasis declares itself varies greatly —that is, from a few weeks to several years.

Thirdly, judging from the text-books, there is a general impression abroad that foreign bodies enter the right bronchus very much more frequently than the left; and several explanations of this supposed fact have been offered, such as the position of the inter-bronchial septum, the greater size of the right bronchus, and the fact that the right bronchus more closely follows the line of the trachea than does the left. If the records of 56 cases which I have collected in the English journals can be relied upon, and are of sufficient number to be of value, this view is ill-founded; because the right side was affected in 32 instances and the left in 24—that is in the proportion of 4 to 3. There is no evidence for supposing that the size of the foreign body has any influence in this connection. An interesting feature is that of 62 cases, 41 were males, and only 31 females.

In conclusion, this short paper will have achieved its main object if fresh attention is drawn to the danger attending the administration of chloroform in dental surgery.

CLINICAL CASES

EXHIBITED.

*** Published in accordance with the regulation relating to the exhibition of patients at the meetings of the Society, viz. that each case shall be accompanied by a brief written description of the points it illustrates; such description to be published or not in the *Transactions* as the Council may direct.

I.—*Traumatic Cephalhydrocele in a Baby æt.* 6 *weeks treated by Operation.* By Douglas Drew, B.S. *Exhibited October* 26, 1906.

THE infant was admitted to the hospital within a few hours of the accident with a large swelling upon the right side of the head.

The history given was that it had fallen off the bed. There was no loss of consciousness. For two days after admission twitching was noticed in the left arm and leg, and there was distinct impulse in the swelling when the child cried. The tumour gradually increased in size, and the gap in the bone, which could be felt by pressing through the swelling, became much wider—*i. e.* about half an inch.

Owing to the increasing size and the unfavourable prognosis in such cases, operation was decided on and performed on the sixteenth day. The object in view was to close the rent in the dura and draw the bones together.

At the operation the sac containing the cerebro-spinal fluid was found to be formed of the separated periosteum. On allowing the fluid to escape profound shock ensued.

The fracture was 1½ in. long, situated horizontally in the parietal, and measured ½ in. across, and through the centre of the opening a hernia cerebri protruded.

Owing to the condition of the child no attempt was made to suture the dura; both the bones were forcibly drawn together with silver wire, the hernia cerebri cut off, and the periosteum replaced and scalp wound sutured.

No unfavourable symptoms followed the operation, beyond slight rigidity in the left arm, nystagmus, and deviation of the eyes to the right, which lasted four days.

II.—*A Case of Traumatic Separation ? of the Head of the Femur.* By Charters J. Symonds, M.S. *Exhibited October* 26, 1906. With Plate XIV.

G. B. was first admitted into Guy's Hospital on October 1, 1904, under me, complaining of some pain about the left hip. He stated that in the spring of 1904 he fell from a tree while bird nesting. He limped home with difficulty, and was quiet for a few days. He resumed his work, and after about three months he began to have rheumatic pains in the hip. He was obliged to give up his work, and then went to the London Hospital, where he attended as an out-patient, and wore a splint for six months. He does not seem to have been any better, and then entered the Hertford Infirmary, where he remained in bed three weeks, and was discharged wearing another splint. He came in under me again on February 26, 1906. At that time the condition was much the same as exists at the present. On looking at the hip the trochanter is seen to be prominent, and to be raised nearer the iliac crest. Part of the prominence is no doubt due to the wasting of the muscles. The hip can be flexed to 45 degrees without pain, and the movements are smooth, showing the articulation itself to be healthy. The movement of abduction and rotation outwards are limited, and when pressed cause him pain. The limb lies in a position of outward rotation, and there is no lordosis. The shortening, which amounts to $1\frac{1}{8}$ in. (the respective measurements being $33\frac{3}{4}$ and $34\frac{7}{8}$ in.), is accounted for entirely by a change in the neck of the femur. In February I took the view that it was a case of tuberculous epiphysitis, and that as he was able to walk with very little inconvenience he might continue to do so, as the local con-

dition seemed to have recovered. He came back, however, complaining of accession of pain a few days ago.

The skiagram (Pl. XIV) taken within the last few days is exactly similar to the one taken in February. It shows that the head of the bone lies in the acetabulum; the neck appears to be thickened and somewhat irregular, and the appearance suggests that the neck has slipped up, so as to leave the lower margin of the epiphysial surface of the head exposed. It is this appearance that has suggested a separation of the epiphysis. The appearance of the neck, however, suggests that some chronic change has taken place more of the nature of a chronic ostitis, due to tubercle, than what would result from an injury. I am, therefore, disposed to leave the diagnosis open at the present time between an epiphysitis—the result of tubercle—and traumatic separation of the neck of the femur. Somewhat in favour of the injury of the traumatic view is the outward rotation of the limb. Comparing the skiagram with those of undoubted fractures of the neck of the femur, the resemblance is, in many respects, very striking. Not having in my possession a radiograph of a recent separation of the epiphysis I am unable to institute a correct comparison; but by placing the case on record, I hope it will enable others to arrive at a conclusion as to whether this is a traumatic condition or not.

III.—*Malunited Fracture of both Bones at the Ankle-joint.* By W. Watson Cheyne, F.R.S. *Exhibited October* 26, 1906.

G. B., fell on July 25, 1903, and twisted his left leg under him. The result was a bad fracture through base of internal malleolus and the fibula at a higher level. Treated at an infirmary for about nine weeks.

January 5, 1904.—Admitted to King's College Hospital with very marked angular deformity, the leg being turned inwards just above the ankle, forming an angle of 150° with the leg above it. The foot is also rotated inwards, and the patient walks on the outer border of the foot. Shortening of left leg to extent of 1 in. and slight wasting of calf muscles.

PLATE XIV.

Illustrating Mr. Charters Symonds' Case of Traumatic Separation of the Head of the Femur.

Skiagram to show the affected neck of the femur.

Bale and Danielsson, Ltd.

January 13, 1904.—Two lateral incisions made, 5 in. in length, one over each bone. Muscles and periosteum stripped off, and the bones which were fractured obliquely divided along the line of fracture with chisel and hammer. After some portions of new bone had been clipped off the position was corrected and the fragments of the tibia were fixed together by an aluminium plate and tacks. The sharp end of the upper fragment of the fibula was inserted into the medullary cavity of the lower fragment, and thus the bones were kept in firm apposition without any wire, etc. Wound healed well.

IV.—*Case of Deformity of the Long Bones.* By T. H. Kellock, B.C. *Exhibited October* 26, 1906.

THE patient, a girl now æt. 8, is an only child; the parents were of no relation before marriage, but there is a slight history of tuberculosis on both sides; none of any other specific disease. There was a miscarriage soon after marriage, and this child was born subsequently at full term. The labour was difficult on account of the large size of the child, and forceps were used, which are said to have injured the head and face a good deal. The child was nursed and given Robb's biscuits occasionally till nine months of age, after which she seems to have been quite properly fed. When two years old she had an abscess in connection with the lower end of the right humerus; this was opened and scraped but no bone removed, and complete recovery followed. A year later she was seen on account of slight genu valgum, and a few months after that for greenstick fracture of the bones of the left forearm. About eighteen months ago she began to suffer from convulsive fits, which occurred about once a month and lasted from ten to fifteen minutes; latterly these have ceased. From about the time the fits commenced the deformity of the legs became worse; she walked but very badly, and had a habit of sitting with the feet turned up under the thighs.

About a year ago the left radius and ulna were again broken as the result of a fall from a chair. From September, 1904, great care has been taken in the matter of diet, and

various drugs, including thyroid extract, have been used without obvious benefit.

The deformity of the bones of the legs is now very great; there is very marked genu valgum, and the feet are strongly inverted as the result of curvature outwards at the lower ends of the tibiæ and fibulæ; the femora are a good deal curved in a forward and outward direction.

In the upper limbs there is bending about the middle of the radius and ulna on both sides, although there is no history of fracture on the right side. At the upper ends of the humeri a marked thickening and anterior curvature is noticeable.

The general health is good and the mental faculties normal.

Skiagrams of the bones are shown, one of the leg taken a year ago, and others of the knee, ankle, forearm, and shoulder taken recently. They all show that the chief part of the deformity is apparently due to fractures and irregular ossification occurring at short distances from the epiphysial lines.

V.—*Case of Symmetrical Affection of the Hip Joints.* By T. H. Kellock, B.C. *Exhibited October* 26, 1906.

M. M., boy, æt. 8, the third member of the family. The eldest died at the age of three months of a convulsive fit, the second is alive and well, as is also the fourth, a girl now six years of age.

There is a history of tuberculosis on the mother's side; the father is a healthy man forty-four years of age.

The patient was born at full term; when seven weeks old he had a severe illness which was said to be dysentery, after which the right knee became very swollen and remained so for about two months, when it got quite well. He did not walk until eighteen months of age, and was very late in cutting the temporary teeth.

About a year ago it was first noticed that his gait was peculiar, but he has never complained of any pain in the hips and has not been laid up.

He has several times suffered from "weak eyes," but has no apparent defect of vision.

Now he has a peculiar gait rather resembling that in congenital dislocations; the trochanters, especially the right, are raised and appear prominent when the legs are adducted. There is no pain or tenderness, and the movements of the joints are free.

A skiagram shows considerable destruction of the head of the femur and an outgrowth of bone round the acetabulum on both sides.

VI.—*A Case of Partial Heart-block.* By PERCY NORTHCOTE, M.B., and A. M. GOSSAGE, M.D. *Exhibited October* 26, 1906.

THE patient, a clerk, æt. 58, was first seen in January, 1906. He had had syphilis at the age of twenty-one, but had otherwise been healthy until four years before, when he had a sudden attack of unconsciousness, falling and injuring his head. Since then he has had numerous similar attacks, more frequent lately. The attacks are either a momentary giddiness, with or without falling, or at other times complete unconsciousness for several seconds, during which time his head is said to roll and his limbs to twitch. He had been aware for about four months before he came under observation that his pulse was beating at the rate of about 30 per minute. During his illness he has felt the cold very severely.

On examination the arteries were found to be much thickened and the tension decidedly increased, the sphygmomanometer showing that the systolic blood-pressure was 178 mm. of mercury. The urine generally contained a small quantity of albumen. Nothing abnormal was found in the heart except the rhythm. When first seen the ventricles were beating at 68, but the patient suddenly felt slightly giddy, and the rate dropped to 32—*i. e.* to about the half of the former rate. This sudden halving of the pulse-rate was repeated on several occasions, the change being generally but not invariably associated with a feeling of giddiness. As a rule the pulse-rate was between 26 and 36, although occa-

sionally it rose to over 60. On several occasions the patient was kept for some days in bed under observation, and on every occasion he was benefited by the rest. Hard work and worry always caused the attacks to be more numerous. No severe attacks took place while he was under observation, but there were numerous slighter ones, from momentary giddiness to attacks in which he would have fallen if not supported. A large number of tracings were taken from his pulse, but as there was no pulsation of the veins these did not give much information. Observation under the X rays showed, however, that the auricles were beating at double the rate of the ventricles, and the bradycardia was probably due to depression of conductivity in the bundle of His between the auricles and ventricles. The more severe attacks were associated with the non-conduction of a series of auricular beats to the ventricle; for instance, in one attack no sound was heard over the heart for five seconds. In the tracing shown the interval between two successive ventricular beats is also about five seconds. In the still more severe attacks there was probably a still longer interval without a ventricular beat. On no occasion were any intermediate sounds heard between the larger ventricular beats.

VII.—*A Case of (?) Syphilitic Disease in the Bones of One Limb.* By Edred M. Corner, B.C. *Exhibited October* 26, 1906.

THE case was that of a little girl with enlargements to the ends of the long bones of her right leg and the right side of her pelvis. The enlargements were thought to be syphilitic, but were found subsequently to be cartilaginous. As the case was such a remarkable one, its description is deferred to a future date.

VIII.—*Three Cases of Patent Ductus Arteriosus with Arterio-venous Murmur.* By H. G. Turney, M.D. *Exhibited October* 26, 1906.

PATENT ductus arteriosus, continuous murmur at left base with thrill.—E. B., æt. 20. At age of two patient is said to have had a cough and to have spat blood. She was taken to the Westminster Hospital, where the mother was told that the child had heart disease. From that time patient has had various attacks of hæmoptysis. She has had a mild attack of chorea and some vague pains in the joints, but no rheumatic fever.

On examination.—Cheeks highly coloured. No clubbing of fingers. Heart: at left base a harsh, continuous thrill, most marked during systole. With this a murmur, which continues throughout the cardiac cycle.

Case 2.—E. M., æt. 29. Except that she was subject to pains in the joints during the winter, there is nothing of note in the history till the present illness.

Four months before admission she complained of severe pain about the heart and of cough. A month later her cough became more severe and she began to spit blood. Since then she has had epistaxis and bleeding from the gums. Her face was noticed to be blue, and has been so ever since.

On examination.—Face very cyanosed. Fingers clubbed. At the left base a murmur was heard, and a thrill felt similar in character to that described above. There was in addition a systolic murmur at the apex.

Case 3.—B. J., æt. 31. When she was a month old, patient was said to have had scarlet fever. The doctor then examined her chest, and told her mother that her heart had been affected. Since then she has had attacks of cardiac trouble, but has been able to lead an active life in service with only occasional breakdowns.

On examination. — Moderate cyanosis. Fingers very slightly clubbed. Condition of heart as in the other cases. At the pulmonary area a thrill and murmur which extend through the cardiac cycle.

IX.—*Large Abdominal Tumour.* By Sir Hugh Beevor, Bart., M.D. *Exhibited October 26th*, 1906.

ALBERT P., æt. 16. The swelling has some features only of enlarged spleen. Symptoms of mechanical interference only. Blood normal. Condition stationary for twelve months. Dyspnœa first noticed two years ago.

X.—*A Case of Double Coxa Vara.* By R. J. Godlee, M.S. *Exhibited October* 26, 1906.

THE patient is a boy, æt. 16, who states that, though he always turned his toes out, he was active, and could play games—*e.g.* football—up to August, 1905, when a barrow fell against him and broke his right thigh. He was in splints for seven weeks. After they were removed the right lower limb was noticed to be shorter than the left. But he continued his work till August, 1906, when he had to give it up on account of pain in the left hip. He had then to take to crutches.

The right great trochanter is more prominent than the left, and is three quarters of an inch above Nélaton's line. All the muscles of the right lower limb are wasted. Both limbs are rotated out on both sides; abduction, flexion, and rotation in are much limited. On bending the knees the legs cross. Walking is difficult; the gait is that common in cases of coxa vara.

The knee-jerks are exaggerated. There is no sign of spinal disease. The ends of the long bones are rather thick, suggesting rickets. There are no signs of tubercular disease.

Skiagrams appear to show double coxa vara, with possibly fracture of the neck of the right femur.

XI.—*A Case of Xanthoma, with Enlargement of the Liver.* By H. C. SEMON, M.B., (introduced by J. ROSE BRADFORD, M.D., F.R.S.). *Exhibited October* 26, 1906.

THE patient, a widow, æt. 60, was admitted to University College Hospital with the history that for seven years she had had jaundice and languor; the urine had been observed to be dark and the motions pale. Two years ago the eyelids altered in colour and texture, and soon after numerous yellow masses developed on the limbs and body. The liver also was found to be enlarged, and laparotomy was performed in the Sussex County Hospital. The organ was found to be indurated and scarred, and numerous enlarged glands were felt in the abdomen, along the spine, and in the portal fissure.

At the present time the liver is felt to be much enlarged, reaching from the sixth rib in the right nipple line to 1 inch below the umbilicus. The yellow nodules of the skin are in some cases movable, but in others appear to be fixed in a varying degree to subjacent periosteum or bone. Bile is found in the urine. The patient's hair is scanty. Other than a history of two miscarriages there is nothing to report of past illnesses.

XII.—*A Case of Acromegaly.* By H. C. SEMON, M.B. (introduced by J. ROSE BRADFORD, M.D., F.R.S.). *Exhibited October* 26, 1906.

THE patient, a married woman, æt. 27, has suffered for three years with pain in the left side of the face and cranium, occurring in paroxysms. The hands and feet have been gradually increasing in size during the last eight months. There has also been amenorrhœa for three years, and the amount of urine and frequency of micturition are increased.

Besides the alteration of the hands and feet, the nose and tongue are enlarged. There is also well-marked bi-temporal

hemianopsia, and both discs show evidence of optic atrophy. Oblique illumination of the nasal half of the left disc produces only sluggish contraction of the left pupil. The urine shows a trace of albumin occasionally. In other particulars the patient is quite healthy.

XIII.—*Case of Cerebellar Tumour.* By D. C. L. FITZWILLIAMS, C.M., for C. A. BALLANCE, M.S. *Exhibited October* 26, 1906.

AGE at present 6 years and 3 months. Admitted to the Hospital for Sick Children under Dr. Colman, May, 1906.

History.—When eighteen months old it was noticed that the child was weak on the left side chiefly in the arm. Said to be rather deaf on the left side. Three months before admission had bad headaches, principally at the back, but also at the front part of the head. Vomiting came on at the same time, independently of food. Slept night and day between the attacks of pain.

On admission.—The cerebellar region bulged on the left side (see cast). Scar on the right arm thought to be tubercular. Left arm weaker than the right, no difference in the legs. Inco-ordination more marked on the left side. Gait unsteady; did not fall more to one side than to the other. Epigastric reflexes increased on the right side, diminished on the left. Knee-jerks equal. Slight extensor response on the left side.

Eyes.—Slight internal squint; very slight nystagmus on looking to the right. No optic atrophy.

Operation.—By Mr. Ballance, in two stages, June 22 and 24. Left side of the cerebellum exposed, œdema of brain over the site of the tumour, which occupied all the left lobe of the cerebellum extending to the middle line, to the pons below, and attached to the tentorium above. Stood the removal well.

July 3.—Became very ill; vomited seven times in the morning; temp. 104°.

July 20.—Scar quite firm; movements very inco-ordinate; nystagmus marked; no optic neuritis; no extensor response; speech very drawling.

After that date the inco-ordination diminished, and has now disappeared; he can walk about. No bulging of the scar.

Cast of head before operation and the tumour (tubercular) removed are exhibited.

XIV.—*Case of Acromegaly.* By Seymour Taylor, M.D. *Exhibited January* 25, 1907. With Figure.

Lily B., æt. 32, domestic servant, complains of increase in the size of her hands and feet, commencing five years ago. She has had no previous serious illness in her lifetime, and has only suffered occasionally from quinsy. There is no predisposition to giantism in any other member of her family.

The face (see Fig.) is very large, the lower jaw and nose especially enlarged. The angle of the lower jaw is very obtuse, and the lower teeth slightly overlap the upper ones. The hands are enlarged, and the fingers flattened, and the forearms generally have an apparently increased bulk, with muscular hypertrophy. The feet and ankles are also enlarged in a manner similar to the upper extremities.

The ribs and clavicles are also large and prominent, but there is no marked spinal curvature. The tongue is thickened, the lips are pendulous, and the speech leathery in tone.

There is no special clinical feature connected with the skin. The thoracic and abdominal organs are healthy. The pulse is normal; but the temperature falls slightly below the normal line. Her chief discomfort is due to attacks of dyspnœa, especially in lying down at night.

Treatment by pituitary extract has afforded negative results.

Dr. Seymour Taylor's Case of Acromegaly, the hands contrasted with the hand of a nurse.

XV.—*Congenital Heart Disease with Cyanosis.* By F. Parkes Weber, M.D. *Exhibited January* 25, 1907.

THE patient is a rather tall, weakly-developed girl, æt. 17. She is generally more or less cyanosed, but her colour varies greatly from time to time. Sometimes it is almost natural, but at other times (especially on physical exertion, or mental effort, or excitement) her lips, nose, and fingers may become bluish-black. There is scarcely any "clubbing" of the fingers and none of the toes. The *heart* appears to be not enlarged, but there is slight bulging of the cardiac area, probably an early rachitic change. No thrill can be felt. When the patient is lying on her back a harsh systolic murmur can be heard all over the heart, loudest in the third and fourth intercostal spaces to the left of the sternum. The murmur becomes less marked during any temporary tachycardia (*i. e.* from excitement), but the *most striking feature* of the case is that when the patient stands upright the murmur almost completely disappears or becomes much fainter and markedly influenced by the respiratory movements, being best heard during inspiration. The pulse-rate averages 93 to the minute and the blood-pressure is low. Blood: Hæmoglobin (by Haldane's method) 115 per cent.; red cells 6,375,000 in the c.mm.; white cells 13,235; by spectroscopic examination of the blood nothing abnormal can be detected. In March, 1906, the polycythæmia was more than it is at present, the blood-count then showing 7,352,000 red cells in the c.mm. No evidence of disease in any other organ. No dyspnœa. The cyanosis seems first to have been noticed when the patient commenced to walk (at the age of 2¼ years), and since then it has remained, varying much, however, from time to time. Menstruation commenced at fourteen years of age. A sister of the patient, æt. 10, is imbecile and backward in physical development. It would seem that the only way to account for the symptoms is to suppose that there is a considerable aperture in the inter-ventricular septum with fairly equal development of the two sides of the heart. The patient was exhibited at the Medical Society in 1906, as a "Case of Congenital Paroxysmal Cyanosis," but now the variations in the cyanosis are hardly decided enough to warrant the use of the epithet "paroxysmal."

XVI.—*Fracture of Tarsus.* By JOHN R. LUNN, F.R.C.S. *Exhibited January* 25, 1907.

THE patient, a porter, single, æt. 22, was admitted into the Marylebone Infirmary, W., on May 2, 1906, with a large swelling of his left ankle. Some doubtful crepitus could be felt. He stated that he had jumped a depth of fifteen feet, landing on his left foot. The X rays revealed a fracture of the astragalus, and a piece of the bone was projecting on the dorsum of the foot. The foot and ankle were put up in a plaster-of-Paris splint, which was removed on June 10, when there was good movement of the ankle. The patient was discharged on July 15 walking well. He was last seen by me in November last, when he told me that he was doing his work as porter.

XVII.—*Achondroplasia.* By JOHN R. LUNN, F.R.C.S. *Exhibited January* 25, 1907.

JAMES G., æt. 53, male, labourer, admitted June 4, 1906, into St. Marylebone Infirmary, W. Patient admitted complaining of sciatica.

Family history.—Father (6 ft. 2 in.) mother (5 ft. 10 in.), three brothers, one sister, all in good health.

Previous history.—Patient has been stunted from birth. He lost the use of his limbs at the age of three years for a time; he was taken to the Orthopædic Hospital, where he was given splints, which he wore for six months. Since this time his legs have been bowed. At the age of seventeen or eighteen he became a coal-porter, which occupation he followed for nine years. Since giving up this employment he has been a labourer on the Paddington roads. Except for occasional bilious attacks he has always enjoyed good health.

History of present illness.—Eight years ago he began to suffer from recurring attacks of sciatica, which eventually compelled him to give up his work. Intellect good. Height 4 ft. 1 in., when sitting 2 ft. 9 in. Circumference of head 24 in.

Measurement of certain bones.—Clavicle 5½ in.; acromion

process to external condyle 6¾ in.; radius 6¾ in.; olecranon to styloid process of ulna 8 in.; anterior superior spine to internal malleolus 18¼ in. on right side, 18½ in. on left; internal tuberosity of tibia to internal condyle 7½ in.; chest, full expansion 35 in.

Appearance.—The forehead is prominent, the bridge of the nose rather deficient, the eyes deeply set. The teeth are ground down, carious, with many teeth missing. The palate arch is rather high. The tongue normal. The legs are very bowed, there being great curvature of the tibiæ. The feet are short and square, with all the toes nearly the same length. There is very little curvative of the bones of the upper limbs. The fingers are more nearly the same length than normal. The middle and ring fingers diverge at the first interphalangeal joint; this is more marked on the left hand than the right. There is lordosis and prominence of the abdomen. The umbilicus lies midway between the crown of the head and the soles of the feet. The finger tips reach an inch and a half below the great trochanters. The genital organs are normal.

XVIII.—*Unusual Case of Enlargement of the Head in a Child.* By Bertram Abrahams, M.B. *Exhibited January* 25, 1907.

C. B., æt. 3. Before his birth mother suffered from what was possibly syphilis. Labour normal. Nothing wrong noticed with head till the age of eight months. The head then began to swell, and continued to do so for some four months, since when it is said not to have increased. Dilatation of the vessels at the root of the nose was first noticed at eighteen months, and has progressed since; before that time the mother "thinks there was a red flush on the forehead."

Present condition.—An intelligent and well-developed child. Head is very large, bulging particularly above and to the back. Greatest circumference 25 in.; length from root of nose to occipital protuberance 15 in.; width between ears (over vertex) 14 in. Percussion note over heart clanging. On auscultation a systolic bruit is heard all over the heart.

The right fronto-parietal fissure is closed, but the left, both coronals, and the frontal portion of the sagittal are

patent. The left occipito-parietal region is larger than the right.

There is venous dilatation over the glabella, involving the left upper eyelid, and descending to within three quarters of an inch of the tip of the nose. The appearance simulates that of a cirsoid aneurysm.

There is no other abnormal physical sign excepting a systolic bruit over the whole præcordia, loudest in the mitral area, and conducted into the axilla.

XIX.—*Successful Plastic Operation for a Syphilitic Perforation of the Hard Palate on the Left Side the size of a Sixpence.* By H. L. BARNARD, M.S. *Exhibited January* 25, 1907.

FRANCIS P., æt. 28, had syphilis four years ago. Perforation present six months.

August 16, 1906.—An L-shaped flap was turned in from the right side of the palate and sutured to the freshened edges of the perforation. The flap was about three times the area of the perforation.

After-treatment.—Potassium iodide and Liq. hyd. perchlor. and frequent mouth-washes of weak carbolic. Healing occurred normally, but as the flap contracted the anterior half of the perforation opened.

December 9.—The entire left half of the palate raised as one flap and sutured over the perforation.

Progress.—Normal healing under the same treatment and the perforation is now closed. A minute perforation in the soft palate, which causes little trouble, remains.

XX.—*Removal of a Stone from each Ureter for Double Pyonephrosis.* By H. L. BARNARD, M.S. *Exhibited January* 25, 1907.

MARY B., æt. 41, had renal colic on both sides, but chiefly on the left, for four years. Attacks of cystitis and pyrexia for three years.

Both kidneys were enlarged, movable, and tender, especially the left. Great wasting. Weight 7 st. 1 lb. Œdema of the feet. Urine = 1010—1016, acid, turbid, albumen, much pus. Radiographic report: (1) A stone the shape and size of an almond in the right ureter opposite the fourth lumbar vertebra; (2) another, smaller, in the pelvis, opposite the fourth sacral. The latter could be felt in the left fornix of the vagina.

October 14, 1904.—The right ureteric calculus was removed through a loin incision. Wound in ureter closed with No. 1 catgut; drainage; no leakage of urine.

December 5.—The left ureteric calculus was removed through an incision in the left fornix of the vagina. Ureter not sutured. Tube in broad ligament for three days. Urinary fistula closed in ten days.

January 25, 1907: *Present condition.*—Well and strong. Weighs 9 st. 10½ lb. A little pain at times in left kidney, which is still somewhat enlarged and tender. Urine slightly turbid, contains pus, acid, 1016, trace of albumen.

XXI.—*A Method of Diminishing the Raw Surface after Removal of half the Tongue.* By H. L. Barnard, M.S. *Exhibited January* 25, 1907.

MARY H., æt. 55, admitted to the London Hospital, May 10, 1906, with epithelioma of the middle of the right edge of tongue due to a sharp carious tooth. No glands felt.

May 14, 1906.—Right half of tongue removed by Whitehead's method, and the submaxillary and deep cervical glands cleaned out. The half of the tongue remaining was doubled back so that the tip was brought in contact with the posterior third. Three catgut sutures inserted.

Advantages of method.—(1) Rapid healing and diminished raw surface; (2) smooth and rounded edge to tongue; (3) small scar.

Disadvantages.—(1) Local recurrence, if it occurred, would be in the middle of tongue; (2) the sensation of the tip of tongue being down the throat, but it has now moved round to the front.

XXII.—*Multiple Chondromata of Metatarsal Bones, with unusual changes in one (?) as a result of Traumatism.* By W. F. Brook, F.R.C.S. *Exhibited January* 25, 1907. With Plate XV.

MALE, æt. 50. While wrestling fifteen years ago right "great toe joint" was badly crushed by opponent's heel. This was followed by occasional painless swelling, which, in the course of a few years, became permanent, but caused no trouble until the beginning of 1907, when it became necessary to wear a specially made boot on that foot.

When first seen in February, 1904, he presented a painless swelling about the first metatarsal bone, which pushed the second toe and metatarsal outwards. Movement of the toe was slightly impaired, but caused no pain or grating. Under the skin of the first interspace, and on either side of the extensor tendon, could be felt freely movable fragments of bone or cartilage of varying shape and size. These fragments appeared to be the main cause of the enlargement.

The swelling is now considerably larger and more tense and the interspace further widened. The fragments are less freely movable, and protrude into the web, whence one was recently removed for examination. The joint is almost rigid, being apparently fixed by the increased number of fragments as by a splint. The slight lameness and pain after use from which he now suffers appear to be due entirely to this rigidity.

All the points shown in a skiagram taken in February, 1904, viz. the breaking up of the head, and, to a less extent, of the shaft of the first metatarsal and base of the phalanx, are more pronounced in the skiagram taken in January, 1907, but especially to be noted is the increase in the collection of cartilaginous fragments undergoing ossification in its centre, with further widening of the interspace and distortion from pressure of the second metatarsal. Moreover, chondromata have appeared in the shaft of the third and the head of fourth metatarsal.

The fragment removed was not encapsuled, and was kindly examined by Mr. Shattock, who reports that it "consists of hyaline cartilage in which small areas of mucus metaplasia occur, *i. e.* areas in which the matrix has become liquified,

PLATE XV.

Illustrating Mr. W. F. Brook's Case of Multiple Chondromata of the Metatarsal Bones.

The diseased right foot is shown in comparison with the normal left foot in a photograph and also in a skiagram.

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PLATE XVI.

Illustrating Mr. Clutton's Case of Congenital Hypertrophy or Giant Growth of Feet.

Photograph of patient.

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PLATE XVII.

Illustrating Mr. Clutton's Case of Congenital Hypertrophy or Giant Growth of Feet.

Skiagram of right foot.

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PLATE XVIII.

Illustrating Mr. Clutton's Case of Congenital Hypertrophy or Giant Growth of Feet.

Skiagram of left foot.

and the cells have become branched to produce a mucous tissue."

It seems probable that some relation exists between the injury, which was evidently severe, and the present condition. In the light of the recent appearance of chondromata in other metatarsals, a possible explanation is that at the time of the injury the first metatarsal was the seat of a chondroma, that this was crushed, the fragment being extruded through the ruptured periosteum, with the result that they have developed a kind of diffuse chondromatosis in the surrounding tissues.

XXIII.—*Congenital Hypertrophy or Giant Growth of Feet.* By H. H. Clutton, M.C. *Exhibited March* 22, 1907. With Plates XVI, XVII, XVIII.

A GIRL, æt. 17, was shown with congenital hypertrophy of both feet and of the whole of the left lower extremity. The deformity, which was noticed by the mother at birth, slowly increased up to the age of fourteen. During the last three years the growth has been much more rapid, producing particularly a greater inequality in the length of the two lower extremities. The feet (Plate XVI) are enormous, but fairly equal in size, the distal parts being rather larger than the proximal. The radiographs of the right foot (Plate XVII) shows that the second, third, fourth, and fifth toes, with their corresponding metatarsal bones, are longer and larger than the first, whilst in the left (Plate XVII) it will be seen that the first, second, and third toes, with their metatarsal bones, are more developed. The tarsal bones are much larger than normal, although not quite so exaggerated as the more distal bones. The only exception is the astragalus, which looks as if it were of about the normal size. The soft parts over the bones are also distinctly hypertrophied. The skin is thicker, especially on the soles of the feet, although she has not walked much. The subcutaneous tissue is more voluminous than is natural, and on the dorsum of each foot there is a swelling which resembles a diffuse lipoma. The condition of the skin and subcutaneous tissue does not, however, resemble that seen in elephantiasis. The lower limbs are unequal in length and size. This is mainly

due to an hypertrophy of the left lower extremity, which is larger and longer than the right, but there is not so much difference between the two thighs as there is between the two legs. The difference in length is over three inches in the recumbent position, when measuring from the anterior superior spine to the internal malleolus. But in the erect position a book of two inches in thickness, placed under the right foot, brings the pelvis level.

The radiograph shows the left leg bones to be longer and larger than the right, and of the two the fibula appears to be longer than the tibia, if one may judge by the curvature of this bone behind the tibia.

There are no other deformities than those above described. The skiagrams were kindly taken by Dr. Greg, and the photos by Mr. G. B. Wainwright. The history of the case was obtained by Mr. Graham Jones.

Note.—The left foot was removed in March, 1907, and has been preserved in the Museum of the Royal College of Surgeons. The right foot will be dealt with in a similar manner during the autumn.—*July*, 1907.

XXIV.—*Case of Pancreatic Fistula.* By F. W. Forbes-Ross, C.M. *Exhibited March* 22, 1907.

W. C., male, æt. 71. The patient had undergone an operation for the removal of piles a fortnight before, when, on October 17, 1906, whilst straining at stool, he suddenly cried out, owing to pain in the left hypochondrium. Signs of septic inflammation set in and a swelling appeared, which increased until it distended the left half of the abdomen as far as a line drawn from the left anterior superior iliac spine obliquely upwards to the right hypochondrium. Mr. Pearce Gould was then consulted; he diagnosed an abdominal abscess, and advised incision.

On October 24 Mr. Ross made an ilio-lumbar incision through the abdominal wall, pushed aside the peritoneum and opened a cavity containing two and a half quarts of sanious inodorous pus. A large drainage-tube was inserted, and the rest of the wound closed. The fluid discharged gave the reactions of the pancreatic secretion. There is now a sinus

3½ inches in depth, discharging a small amount of fluid. The patient has recovered health, and has returned to his business. A pancreatic cyst may have been slowly forming, in which suppuration set in a week before the incision.

XXV.—"*Pulmonary Hypertrophic Osteo-arthropathy*" *occurring in a case of Congenital Heart Disease* (*!*).

By H. Batty Shaw, M.D, and R. Higham Cooper, L.S.A. *Exhibited March* 22, 1907. With Figures.

THE patient, a young unmarried woman, æt. 21 years, was recently admitted to University College Hospital under the care of Sir Thomas Barlow, to whom we are indebted for permission to publish the notes of the case. The patient had suffered from palpitation, breathlessness, and blueness since birth. During the last ten years she had suffered from pains in the limbs, both in the ankles and wrists and in the shafts of the long bones of the legs, more particularly after standing and walking. She had scarlet fever at the age of ten years, but no other illness. There was no history of acute rheumatism or of syphilis in the family, the other members of which were quite healthy. The patient was very cyanosed, and clubbing of the fingers, the toes, and the nose was well marked. A systolic murmur was heard over the præcordium, probably due to defect of the interventricular septum; it was best heard to the left of the sternum and in the third and fourth intercostal spaces, but was also audible at the apex beat of the heart and in the second left intercostal space; it was not conducted towards the left clavicle. There were no signs of any of those diseases of the lungs which have hitherto been found associated with clubbing of the fingers and osteo-arthropathy; the tuberculo-opsonic indices observed on five separate occasions by Mr. D. Embleton were 0·96, 0·78, 0·96, 0·96, and 0·82. The blood examination showed 8,320,000 red corpuscles and 9300 white corpuscles per cubic millimetre; the percentage of hæmoglobin was 150, and the colour index was 0·94; the polymorphonuclear cells numbered 60·2 per cent., the small lymphocytes 19·9 per cent., the large lymphocytes 12 per cent., the hyaline cells 6·9 per cent., and the eosinophile cells 1 per cent. Besides the clubbing above referred

to there was obvious swelling of both legs due to thickening of the lower ends of the tibiæ and fibulæ (Fig. 1), and there

Fig. 1.

was slight swelling of the bones of the forearms, of the wrists, and of the shafts of the metacarpal and phalangeal bones.

Radioscopic examination.—Compared with the corresponding bones of the patient's sister, who was quite healthy and of almost the same height and build, there was very con-

siderable enlargement of the tibiæ and fibulæ of both legs, especially of the lower two thirds, more marked in the right

FIG. 2.

limb; irregular bony deposits were present at the attachment of the interosseous ligaments to the adjacent tibiæ and fibulæ. The upper halves of the shafts of the femora showed increase of the mass of bone, but in a more marked degree, especially in the right leg (Fig. 2); the head, the neck, and

the lower halves of the femora appeared to be normal. The first and second phalanges of the fingers and thumbs, the metacarpal bones, the first and second phalanges of the toes, the metatarsal bones, and the bones of both forearms showed very slight thickening; there was no bony increase of the terminal phalanges of the fingers or toes.

This case is of special interest in several directions. In the first place it confirms the belief that Pierre Marie's sign-complex is not a feature of particular forms of lung disease only, such as bronchiectasis, tuberculosis, empyema, neoplasm, etc., for the patient was quite free from any signs or symptoms of pulmonary disease. The changes in the bones and the joint pains constitute "osteo-arthropathy," which, combined with clubbing, is known under the title given by Pierre Marie * as pulmonary hypertrophic osteo-arthropathy; the latter observer and Bamberger † considered that the genesis of this sign-complex depended on the effects of toxins formed in the lungs; we consider that everything points to the absence of any toxic focus in the lungs of this case; tuberculosis of the lungs, the most common infection in congenital heart disease, may yield no physical signs and yet be present; that this is not so in this case is shown by the existence of normal opsonic indices above quoted—a condition of affairs which could not occur if a tuberculo-toxin were circulating in the system so widely as to produce the above diffuse osteo-arthropathy. A mechanico-toxic cause of clubbing and of osteo-arthropathy has been advanced recently by Béclère ‡ and by Groedel §, and confirmed by an observation by Berent ‖; clubbing of the fingers has long been known to occur in one hand ¶ in cases in which an aneurysm has caused interference with the circulation of the limb, and Groedel and Berent have shown that bone changes may also occur in such cases; the toxic substance or substances must in these cases be generated locally, and be derivations of tissue metabolism. Their retention, owing to

* Pierre Marie, *Revue de Médecine*, 1890, tome x, p. 1.

† Bamberger, *Zeitschrift für Klinische Medicin*, 1891, Band xviii, S. 193.

‡ Béclère : *Semaine Médicale*, 1901, p. 94. *Bulletins et Mémoires de la Société Médicale des Hôpitaux*, 1901, vol. xviii, p. 283. *Gazette des Hôpitaux*, 1904, p. 754.

§ Groedel, *Münchener Medicinische Wochenschrift*, 1906, S. 264.

‖ Berent, *Berliner Klinische Wochenschrift*, 1903, S. 75.

¶ Sir Thomas Smith, *Transactions of the Pathological Society of London*, 1871, vol. xxiii, p. 74. Canton, *ibid.*, 1865, vol. xvii, p. 428. Ogle, *ibid.*, 1858, vol. x, p. 103.

the obstructed circulation, is considered to be responsible for the clubbing and for the bone and joint changes. We consider that this case gives support to the view of Béclère and of Groedel, because the greatest bone change is noticed in the lower extremities, gravity tending to increase the stagnation of the blood in these parts. That mechanical obstruction is in the first place responsible for the change is also shown by the fact that the patient has suffered for as long a period as ten years frequently and immediately from pain in the lower limbs on standing or walking.

This case is finally a unique one, for, so far as we are able to ascertain, there are no other reports in the literature of cases of congenital heart disease being associated with Marie's sign-complex in the absence of lung disease. Bamberger's eighth case was one of congenital heart disease, but was complicated by the presence of tuberculosis, and Thorburn's case (footnote to his paper), though occurring in a boy, is not stated to be due to congenital as opposed to acquired heart disease; the latter association has been established by Bamberger. The distribution of the changes, mainly in the lower extremities of our case, is also very unusual, and, as already stated, in the absence of any focus of disease in the lung, supports the view that clubbing and osteo-arthropathy are due to the effects of tissue toxins normally generated at the periphery but prevented from being rapidly removed owing to the impairment of the circulation, in our case dependent upon congenital heart disease, in others upon the existence of lung disease, of acquired heart disease, and even, it is stated, of impaired action of the diaphragm in cases of cirrhosis of the liver and also in cases of chronic jaundice.

XXVI.—*Case of Osteogenesis Imperfecta (? Syphilitic).* By G. A. Sutherland, M.D. *Exhibited March* 22, 1907. With Plate XIX.

A MALE infant, æt. 8 weeks, a first child. He was born after an easy labour, and "the left wrist was seen to be broken and the arm to hang as if dislocated. The thigh and leg bones on the left side were broken." Multiple fractures

in the long bones are shown in a skiagram taken seven days later. No fresh fractures have been observed to occur. He seemed to suffer great pain whenever his limbs were touched, and cried for hours after he was born. At birth also a lump was noticed on the right side of the head. There is nothing of importance in the family history.

On admission, four weeks ago, great pain and tenderness were present in the extremities, which were the seat of periosteal swelling, especially round the epiphyses. A condition of pseudo-paralysis existed, movement being limited to the fingers and toes. Signs of fracture were manifest in all the long bones of the extremities, and in many of the ribs (*vide* skiagram [Pl. XIX] by Dr. Jackson Taylor). A swelling was present over the right parietal bone with a large irregular gap in the osseous tissue. There was general peeling of the skin over the trunk and extremities.

Under observation the pain and tenderness in the limbs have passed off, the periosteal swelling has subsided, and the infant now moves the limbs freely. Marked deformities have resulted from the fractures, which are now apparently united. Mercurial treatment has been carried out.

XXVII.—*A Case of Facio-hypoglossal Anastomosis for Post-operative Paralysis; nearly Complete Recovery.* By A. H. Tubby, M.S. *Exhibited March* 22, 1907. With Figures.

J. P. B., æt. 19 years, was admitted into Westminster Hospital on October 3, 1905. While in the Militia, during the early part of that year, a swelling appeared behind the left side of the jaw. This was opened by a surgeon on April 27, 1905, and when he recovered from the operation he found the left side of his face paralysed. When he was admitted to Westminster Hospital it was noted that the left half of the mouth dropped; the left palpebral fissure was larger than the right; the left side of the forehead was unwrinkled; the cheek was flattened, the tongue deviated slightly to the right; the palate moved normally, the uvula being in the middle line, but food accumulated in the left cheek. There were also epiphora and dribbling of saliva on the left side. He

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PLATE XIX.

Illustrating Dr. Sutherland's Case of Osteogenesis Imperfecta.

Skiagram to show the bones of the extremities.

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could not whistle nor draw in the cheek. The appearances are seen in the photograph (Fig. 1). The sensation of touch was not diminished, but he had lost all taste on the anterior part of the left side of the tongue.

On October 16 an anastomosis was carried out. An incision was made from the tip of the mastoid process to the

FIG. 1.—Photograph of the patient before operation.

greater cornu of the hyoid bone, and then a dissection was undertaken to find the cut distal ends of the facial nerve. This was a matter of considerable difficulty. A further dissection was then made to expose the hypoglossal nerve. A large part of the digastric and of the stylo-hyoid muscles was removed, the hypoglossal nerve freed, and brought upwards as near to the facial as possible. An incision was made with a tenotomy knife deeply into the hypoglossal nerve, and the distal ends of the facial were implanted into it laterally. Great care was taken to keep the head quiet, to

prevent all movement of the neck for fourteen days after the operation.

On October 23—that is a week after the operation—it was noted that the palpebral fissure was lessened in size, and the eye could be temporarily closed.

On October 28 the patient began to move the left angle of the mouth.

FIG. 2.—Photograph four months after the operation.

On February 7, 1906, the following conditions of voluntary movement were to be seen : Action of the corrugator supercilii in wrinkling the forehead ; firm action of the orbicularis palpebrarum in the closure of the eye on the left side with control over the muscles of the cheek. He could also voluntarily draw the left angle of the mouth upwards to a higher degree than the right when the latter was at rest, but in repose the left angle slightly drooped. The left ala nasi moved simultaneously with the mouth, and there was flickering of the muscle of the chin on that side. The appearances after the

operation are seen in Fig. 2. It was striking to observe that the tongue was drawn over to the right side, and there was a little thickness of speech. The patient said that when he moved his face he was not compelled to move his tongue at the same time. There was some wasting of the left side of the tongue. When both sides of the face were thrown into action the right side still overpowered the left. He could whistle and the food no longer accumulated in the cheek.

March 22, 1907.—At this date the improvement is fully maintained in all respects. There is less drooping of the left angle of the mouth in repose, while when in action the disparity of movement between the two corners of the mouth is less marked. In fact the improvement has been slightly but steadily progressive.

XXVIII.—*A Case illustrating the Treatment of Complete Paralysis of the Gastrocnemius and Soleus with Talipes Calcaneus, by Nerve-Grafting; Partial Recovery.* By A. H. Tubby, M.S. *Exhibited March*, 22, 1907.

WILLIE T., æt. 9 years, was admitted to Westminster Hospital on May 19, 1905. He was attacked by infantile paralysis in the left leg when aged two and a half years, but recovery took place except in the gastrocnemius and soleus and the right erector spinæ. He was treated at Queen Square Hospital by massage and electricity for three months, and had also worn leg-irons for three years.

On admission to Westminster Hospital it was noted that all the muscles of the left thigh and leg were smaller than those of the right. The patient could extend and flex the knee, the latter by the hamstrings alone, without the aid of the gastrocnemius. He could dorsiflex and evert the ankle. At Westminster Hospital efforts were made to improve the condition by massage and electricity, but no results followed, the gastrocnemius and soleus being found to be useless and paralysed, and not responding either to the galvanic nor faradic currents. The heel was dropped and bulbous, and he could not plantar flex the foot.

On June 5 nerve transplantation was performed. The branches from the internal popliteal nerve to the gastrocnemius and soleus were identified by sterilisable electrodes, and contraction resulting, these branches were separated from the internal popliteal and grafted by lateral implantation on to the external popliteal.

This operation is easy to perform as the nerves to the gastrocnemius and soleus can be traced up for a long way in the internal popliteal as separate bundles, so that an ample length of them can be obtained to bridge over the gap between the internal and external popliteal. The nerve to the external head of the gastrocnemius passes directly to the muscle. The nerve to the internal head gives off branches to the soleus.

No effect could be observed until October 1, when it was seen that some voluntary plantar flexion of the ankle had appeared, but he could not move the foot downwards beyond the right angle. That is, four and a half months elapsed before any return of power could be noted.

Since then the power has increased, and he can plantar-flex the foot to the right angle, but when he walks the action of the gastrocnemius and soleus in lifting the heel is not entirely satisfactory. Up to February 9, 1906, no reaction to galvanism or faradism had appeared in the gastrocnemius and soleus. On May 31, 1906, faradism gave no reaction, but with galvanism there is a faint response, A.C.C. > K.C.C. On March 22, 1907, he reacts well to faradism, and to galvanism K.C.C. > A.C.C. He raises the left heel well in walking, but cannot lift the body weight on the left foot. He can plantar-flex the foot, and the Achilles tendon is felt to harden in doing so.

XXIX.—*Complete Paralysis of the Gastrocnemius and Soleus; Talipes Calcaneus; Nerve-grafting; much Recovery of Power.* By A. H. TUBBY, M.S. *Exhibited March* 22, 1907.

FRANK R., æt. 9 years, was admitted to Westminster Hospital on October 21, 1905, suffering from talipes calcaneus of seven years' duration. He had been in the

West End Hospital for Paralysis for two months, undergoing massage and electricity, but had not improved.

The foot was in a position of marked talipes calcaneus, with great contraction of the plantar fascia. All the muscles of the left leg below the knee reacted to faradism excepting the gastrocnemius and soleus, while to galvanism in the latter muscles the anodal closing contraction is greater than the cathodal, but the contraction is very slight. He had some degree of plantar flexion due to the action of the deep muscles of the leg.

On October 30 the plantar fascia was divided, and the sole of the foot rectified. On November 13 an incision was made, the nerves to the gastrocnemius and soleus, proceeding from the internal popliteal, were identified by using sterilisable electrodes. The nerves were severed from the internal popliteal, and inserted by lateral implantation into the external.

On December 23 it was noted that the tendo-Achillis showed a slight flicker of movement and some tightening in plantar flexion. On January 9 the electrical tests showed that there was no reaction to faradism, and very little to galvanism, A.C.C. > K.C.C. When the foot was dorsiflexed, and some resistance was opposed, the tendo-Achillis could be distinctly felt to harden. On January 31, 1906, the voluntary movement of the heel upwards was more marked. On May 9 it was noticed that he could flex and extend the left ankle well when the foot was off the ground, and the electrical reactions showed that all the muscles of the limb excepting the gastrocnemius and soleus reacted well to faradism, while to galvanism the gastrocnemius gave the reaction of K.C.C. > A.C.C.

On March 13, 1907, it was found that the gastrocnemius and soleus reacted to the faradic current, and with the galvanic current, K.C.C. was > A.C.C. But he has not regained the power of raising the body on the left foot, yet he lifts the heel in walking. When the foot is off the ground he can plantar-flex it to the fullest extent, and the range of movement has become normal since the nerve transplantation was done. When the foot is plantar-flexed it requires a considerable effort on the part of the surgeon to overcome the contraction. It will be noted that in this case power had commenced to return within nine weeks of the operation, whereas four and a half months elapsed in the case of W. T. Further, on January 9, 1906, there was no

reaction to faradism in the affected muscles, while power had begun to return, as evidenced by the movement of the muscles and hardening of the tendo-Achillis. This shows that during recovery the only reliable tests are return of muscular movement and visible tightening of the tendon, while too much reliance as to prognosis must not be placed upon the electrical reactions.

XXX.—*Acute Thyroid Swelling, with Acute Hæmorrhagic Œdema of the Right Side of the Pharynx and Larynx.* By F. PARKES WEBER, M.D. *Exhibited March* 22, 1907.

THE patient, a well-built man æt. 54, was first seen in November, 1906, when he complained of headache and nervous feelings. He was found to have slight cardiac hypertrophy, high arterial blood-pressure (on one occasion up to 230 mm. mercury, as estimated on the arm with the Riva-Rocci apparatus, using the broad band), a small retinal hæmorrhage in the right eye, and a trace of albumen with a few cell-casts in the urine; all this pointing to the presence of chronic interstitial nephritis. No history of syphilis. Within the past two years he has on three occasions (last time, end of January, 1907) suffered from slight temporary aphasia. At the beginning of March, 1907, he had headache and felt unwell (Dr. J. R. Halliday, who attended him, found that the temperature on one occasion reached 100° F.), and was conscious of a pricking sensation on the right side of the neck. On the afternoon of March 4 (about 4.30 p.m.) his neck suddenly began to swell, and he had (subjective) difficulty in breathing. On admission to the German Hospital at 7 p.m. the whole thyroid gland, but especially the right lobe, was much enlarged, tense, and tender. There was likewise considerable general swelling of the neck and over the upper part of the sternum, and there was great œdema of the right side of the pharynx and larynx. The pharynx was scarified, and the patient was much relieved by a morphine injection, rest in bed, and the external application of ice to the neck. A calomel aperient was afterwards given. There was no fever. The urine was of sp. gr. 1026,

containing a little albumen (about one quarter per mille by Esbach's tube), and free from sugar. There was no œdema elsewhere on the trunk or extremities. Next evening (March 5) the swelling of the neck, pharynx, and larynx was already somewhat less, and on March 11, though the thyroid gland, including the isthmus, was still enlarged, the rest of the swelling had subsided, leaving ecchymosis behind it. At present (March 17) the man feels almost in his usual condition again, but the thyroid gland is somewhat enlarged, and the skin of the neck (especially the right side), and the upper front part of the chest is discoloured, as if from extensive bruising. The skin of these parts presents various shades of green, greenish-yellow, and purple, and there is still much ecchymosis of the mucous membrane of the pharynx and larynx.

Blood examination (March 17). — Hæmoglobin (by Haldane's method), 60 per cent. of the ordinary standard; red cells 3,450,000 in the cubic millimetre; white cells 14,700; nothing abnormal by microscopical examination of stained films.

The facts point to the condition having been due to sudden hæmorrhage into the right lobe of the thyroid gland ("apoplexy of the thyroid"), which quickly also distended the isthmus and the left lobe, though to a somewhat lesser degree. The hæmorrhagic œdema of the neck and of the right side of the pharynx and larynx was doubtless analogous to the œdema and widespread appearance of bruising often following bone-fractures and other injuries associated with local extravasation of blood. The swelling reached its maximum about five hours after its commencement.

XXXI.—*Enlargement of the Clavicle and Lower Jaw.* By H. Betham Robinson, M.S. *Exhibited March 22, 1907.*

BOY, æt. 13, with chronic inflammatory swelling of right clavicle and left side of lower jaw. When first seen at end of October clavicle only swollen; this resulted from a fall against a wall one month previously. In November bad toothache and jaw subsequently swelled.

There is some nodular enlargement of right half of thyroid gland. No history of tubercle or syphilis.

There has been some reduction in size under potassium iodide. Latterly he has ceased attending at the hospital and the swellings have increased.

Note.—The swollen clavicle was a few days afterwards cut down upon, and a portion removed gave evidence histologically of chronic inflammation.

Under mercury and iodide of potassium there has been marked improvement.

XXXII.—*Disappearance, more or less complete, of the Subcutaneous Fat above the Region of the Lower Extremities.* By HARRY CAMPBELL, M.D. *Exhibited March* 22, 1907.

GIRL, æt. 21. This is especially marked in the face, chest, and abdomen, including the region of the *mons veneris*. There is no disappearance of fat from the mammary glands or the orbits. There is an abundant deposit of subcutaneous fat in the buttocks and lower extremities generally.

The patient is said to have been perfectly normal up to the age of seven. From this time until the age of fourteen there occurred a gradual disappearance of fat from the affected regions.

XXXIII.—*Patient from whom a Large Polycystic Kidney was removed two and a half years ago.* By DONALD ARMOUR, M.B. *Exhibited March* 22, 1907.

F. S., æt. 29, married, no children, attended the West London Hospital in October, 1904, complaining of persistent frontal headache, pain in the lower part of the back, abdomen and left side, pain and stiffness in the legs, and progressive weakness. There were no symptoms referable to the urinary organs, nor could any history of hæmaturia or polyuria be obtained. She was a pale, anæmic woman, with a dull muddy complexion and a tired, worn expression.

On examination, a large oval tumour was found occupying the left renal region. The patient was quite unaware of its presence. It was slightly movable on palpation, and moved with respiration, dull on percussion except over the anterior surface, which was resonant. There was no tenderness except on deep pressure.

Urine.—Acid, sp. gr. 1024, no albumen, blood, nor sugar. Optic discs normal, no retinitis nor hæmorrhages.

Heart.—Apex-beat in fifth interspace, felt as far out as nipple line; left border half inch outside nipple line; action regular; very faint systolic murmur at apex, not conducted outwards; second sound at base sharp and accentuated. Pulse not of high tension, though the artery felt somewhat thickened.

There was no appreciable alteration in the size of the tumour during eight weeks' observation, but her headache and pain increased.

The kidney (specimen shown) was removed by the usual lumbar incision after the right kidney was found by transperitoneal examination to be normal. Recovery uneventful.

XXXIV.—*Obliteration of Bilateral Psoas Abscess.* By W. G. Spencer, M.S. *Exhibited March* 22, 1907.

FEMALE, æt. 25. At fourteen hurt her back, which pained occasionally afterwards. At twenty-two more pain in lumbar region and down legs.

December, 1905.—First saw doctor.

February, 1906.—Right loin incised, sinus persisted.

October 25.—Thin, weak, pain and slight prominence, lower lumbar vertebræ. Bilateral psoas abscess extending from diaphragm to Poupart's ligament, bulging forwards, tense, the right abscess leaking through sinus.

30.—On right side free extra-peritoneal incision, pus, sloughs, and caseous fibrin wiped out. Wall of cavity thick, pulpy. A sinus leading into a vertebra gouged out. Whole of cavity filled with iodoform gauze. On left side a short incision, cavity washed out, iodoform in glycerin injected, sewn up.

November 20.—Left side had re-filled and become tense, so treated like right side.

Both cavities have been dressed daily with thoroughness, and there is now a granulating hole admitting last joint of little finger. Patient has been up some time, is now stout, and shows no signs of tuberculous disease.

XXXV.—*Case of Fixation of Jaw.* By A. E. Barker, F.R.C.S. *Exhibited March* 22, 1907.

THE patient had been a pupil teacher in a school eight years ago, and was overworked. An attack is described as occurring at that time which is stated to have been accompanied by hemiplegia. The jaw then became fixed, but there had been at the same time some swelling of the left side of the jaw for which teeth were removed. The nervous condition passed off, and has left no traces behind (? hysteria). But the jaw has remained fixed, and the left masseter seems to have become fibrous.

Since the patient was exhibited the jaws have been forcibly separated under chloroform with the greatest difficulty until the incisors were 1¼ in. apart. This was repeated on several occasions, a rubber wedge gag being worn in the intervals. She was sent back to Suffolk able to have her teeth separated but with very little power to move the jaw herself (? hysteria + fibrous change in the muscles after eight years' disuse). Her doctor is carrying out this treatment at her home.

XXXVI.—*Two cases Illustrating the Use of a Filigree of Silver Wire in the Radical Cure of Umbilical Hernia.* By Douglas Drew, B.S. *Exhibited April* 26, 1907.

CASE 1.—F. P., æt. 49, mother of five children. The hernia appeared twenty-five years ago, after the birth of her first child. It was about the size of a cocoanut, and was irreducible.

The recti muscles were widely separated above and below the ring.

The filigree, 8 × 4, was inserted beneath the recti muscles, which were drawn together over it, as also were the edges of the sheath. The operation was performed on February 15, 1907.

Case 2.—E. F., æt. 41, mother of two children. Hernia appeared four years ago, and measured 4 inches in diameter. The filigree, 4½ × 3, was used in the same manner as in Case 1. At the end of a fortnight the wound appeared to be soundly healed, but a few days later some inflammation was noticed at the lower end of the wound, and some broken-down blood-clot and pus were evacuated.

The lower strands of the filigree could be felt with a probe. The wound was fomented, and in ten days it closed, and now appears to be quite firm.

XXXVII.—*Habitual Dislocation of the Patella (? Congenital).* By Douglas Drew, B.S. *Exhibited April* 26, 1907.

L. C., female, æt. 13 years. The child first came under observation when six years old with severe genu valgum (right) and dislocation of the patella when the knee was flexed. McEwen's osteotomy of the femur was performed April 10, 1900, and it was thought that by removing the deformity the tendency to dislocation would be overcome. The child was lost sight of until a fortnight ago. The present condition is much the same as seven years ago. On flexing the knee the patella slips completely over to the outer surface of the external condyle, whereas, when the knee is extended, it lies over the external condyle.

It will be noticed that the muscles of the thigh are smaller than on the left side, and some weakness is complained of.

It is suggested to treat the case by dividing the capsule on the outer side of the patella so as to allow of the reposition of the bone, and by shortening up the capsule on the inner side, and, if necessary, to deepen the patella surface on the femur.

XXXVIII.—*Case of Acute Ataxia (Encephalitis Cerebelli).* By FREDERICK E. BATTEN, M.D. *Exhibited April* 26, 1907.

THIS case was exhibited before the Society in October, 1904.

The boy, æt. 3½, had measles in March, 1904. From this he recovered. On April 2 he vomited, had general convulsions, and passed into a comatose condition. On regaining consciousness he was wildly ataxic both as regards his legs, arms, and trunk.

From this he very slowly recovered, and, when exhibited before the Society on October 28, 1904, was still very ataxic. He is now (April, 1907) six years old. He walks fairly well, but is still unsteady, especially on the left leg. If he be asked to kick a ball the inco-ordination of the left leg is very marked. The articulation is still slow and hesitating. The knee-jerks are present, and the plantars give a flexor response.

Sensation to touch and pain is perfect. He can appreciate the shape of objects put into his hand, and the sense of position is probably good. This latter is, however, very difficult to investigate. His mental condition is fair, but he is backward for his age.

XXXIX.—*Case of Acute Ataxia.* By FREDERICK E. BATTEN, M.D. *Exhibited April* 26, 1907.

F. G., a boy, æt. 11, was quite well up till April 1, when he had headache. On the following morning, after coming downstairs, he fell; he picked himself up, but fell a second time; then he was taken upstairs, and he again fell. He had severe headache and vomited the whole day, and was unable to feed himself. On April 5 he tried to get out of bed, and walked like a drunken man.

He attended the National Hospital on April 9. He had marked ataxia of both legs, some ataxia of the hand, and nystagmus on looking to the left side. There was loss of tone in the muscles of the lower limbs. The knee-jerks were

active, and the plantar reflexes flexor; sensation was perfect to all forms.

The boy rapidly improved, so that he now only exhibits slight ataxia in the lower extremities.

XL.—*A case of Acute Ataxia.* By FREDERICK E. BATTEN, M.D. *Exhibited April* 26, 1907.

E. B., a boy, æt. 12, has always been backward. For the last seven years patient has had attacks of petit mal. In September, 1906, he had a series of 130 fits, and was unconscious for three days. On recovery he could move arms and legs, but had no control over them, and has not been able to walk since. He has had no headache nor vomiting, and has no optic neuritis. He is the fourth of four children; the others are healthy. His mental condition is poor, his articulation is slow and jerky. There is unsteadiness of the trunk and head. There is general weakness of the arms, and a well-marked intention tremor is present. In the legs all movements are possible, but there is marked loss of tone. He cannot walk without aid, and his gait is wildly ataxic. The knee-jerks are active, the plantar gives a flexor response.

Sensation is perfect to all forms, and he has no loss of muscular sense.

There is a lateral curvature in the dorsal region with the convexity to the right. Whilst under treatment he has made but little improvement.

XLI.—*Two Cases of Long-standing Talipes Equino-varus treated by Tibio-calcaneous Resection, with Amputation of the Remainder of the Foot (Le Fort's Amputation modified).* By C. GORDON WATSON, F.R.C.S. *Exhibited April* 26, 1907. With Plate XX.

CASE 1.—Arthur H., æt. 30, no occupation, was admitted to the Metropolitan Hospital, March 31, 1906.

History.—Infantile hemiplegia, æt. 1, leaving partial

paralysis of left hand and leg below the knee. Has had no treatment in the past. Deformity of the foot has gradually increased. Has worn a high boot for past six months (O'Connor extension), but it has constantly given away, and has not given him much support.

He is unable to work as a labourer owing to difficulty in standing. Left arm and leg are much wasted.

The foot is in position of marked equino-varus, with the dorsum directed downwards.

Electrical reactions.

Left thigh normal. The superficial calf muscles react to strong galvanic and Faradic currents. The tibialis posticus and flexor longus digitorum react to very strong Faradism and slightly to very strong galvanism. There is no reaction in the extensors.

The peronei react to strong Faradism and galvanism.

The movements at the hip and knee are good.

The left leg measured to the malleoli is one inch shorter than the right.

Brief details of the operation.—Dorsal skin incision extending from tip of the external malleolus across the head of the astragalus to half an inch below and behind the tip of the internal malleolus. Resection of the astragalus. Removal of cartilage from articular surfaces of external malleolus and inferior surface of tibia, and superior surface of os calcis. Removal of internal malleolus. Plantar flap cut extending from extremities of dorsal to half an inch in front of tubercle of scaphoid internally and to base of five metatarsal externally, and transversely across the sole between these two points. Amputation of remainder of the foot and removal of about half an inch of the anterior extremity of the os calcis. Tenotomy of tendo-Achillis and surrounding contracted tissues. Os calcis pegged with steel pin passed through the sole into the lower end of tibia. Stump put up in plaster. Walked on peg leg for three months, and since then on an artificial foot.

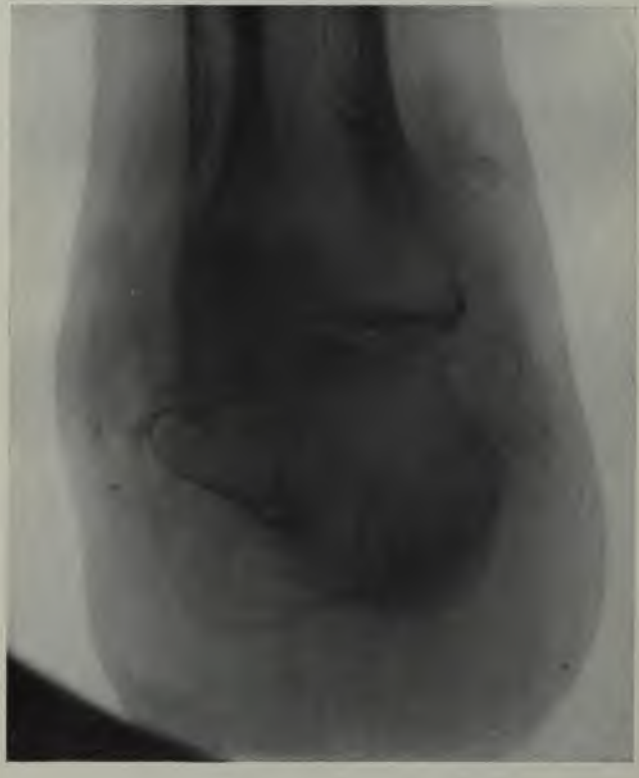
Case 2.—Infantile hemiplegia involving the right arm and leg, and slightly also the left leg. Frank S., æt. 44, admitted to the Metropolitan Hospital, June 28, 1906.

The deformity in this case was almost identical with the first, but the varus was rather less marked. An additional factor in this case was some paresis of the extensors of the opposite leg with rectangular talipes.

PLATE XX.

Illustrating Mr. Gordon Watson's Cases of Talipes Equino-varus.

Skiagram of stump.



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The operation was identical except that rather more of the front of the os calcis was removed as the plantar flap had been cut a little too short.

When the dressings were removed the central portion of the anterior flap was found to be gangrenous, and was cut away. Some blood-clot had collected underneath the flap.

The tourniquet in this case was not removed until after the wound had been closed and firm pressure applied with dressing and bandage.

As there is a certain amount of slack under the anterior flap this is probably an unwise proceeding in these cases. Subsequent skin grafting produced a good scar, but a little depressed in the centre, and not free as in the first case.

The patient commenced to walk on a peg leg five weeks after the operation, when bony union was firm, and the wound soundly healed.

He began to walk on an artificial foot seven months after the operation.

XLII.—*A case of Adolescent Coxa Vara (Traumatic).* By C. GORDON WATSON, F.R.C.S. *Exhibited April 26, 1907.*

A BOY, æt. 16, who came to the hospital, May 4, 1906, with well-marked unilateral coxa vara three months after slipping downstairs.

The accident caused little inconvenience at the time, but gradually since then the symptoms have come on—stiffness, lameness, and shortening.

Skiagram showed separation of head of femur.

April, 1907.—Skiagram now shows transition from "separation of epiphysis" to "typical coxa vara deformity."

Skiagrams and notes shown with patient.

XLIII.—*A Case of Encephalitis chiefly affecting the Mid-Brain.* By Reginald Miller, M.B. *Exhibited April* 26, 1907.

CHILD (male), æt. 3 years, under the care of Dr. Colman, at the Hospital for Sick Children, Great Ormond Street. In October last, child was suddenly taken ill with a convulsion, in which the left side of the face was drawn up, and this was followed by three weeks' unconsciousness. When seen in November there was some weakness of the right side of the face and the right external rectus with slight lateral nystagmus. More marked signs were inability to walk or talk, extreme emotionalism, some rigidity of the right arm and leg, and tremor (about 5 per second) of the head and limbs, which showed more in the proximal than in the peripheral parts of the limbs and was absent only during sleep. The fundi were normal, the abdominal reflexes absent on the right side and feeble on the left, the knee-jerks brisk and about equal, and the plantar responses flexor. The facial and ocular signs quickly cleared up and the tremor became of the "intention" type; the abdominal reflexes became normal, but the rigidity of the right side remains. He is still emotional, nervous, and greedy, although much improved; he has regained the power to walk, but cannot yet speak intelligibly. Recovery has been slow but continuous.

XLIV.—*Lymphangioma of Tongue in a Boy æt. Five Years.* By W. G. Stone, M.D., and F. Norman, M.D. *Exhibited April* 26, 1907.

THE right side of the tongue was severely bitten three years ago. Since then it is reported that there has always been a swelling under the tongue about the size of a hazel nut, with a smooth surface, and of the same colour as the rest of the tongue. On April 19 he fell, and, perhaps, bit the part, which bled, and has since discharged. The present state of the swelling was observed the next day, and the size of it has increased since. There is now an irregular

purple swelling on the under surface of the right half of the tongue with hardened edges. The glands in the submaxillary region are enlarged on the right side, but not tender.

The anterior portion of the right half of the tongue was excised by Mr. W. G. Spencer in the Westminster Hospital, and the tip of the remaining half brought round and fixed by sutures. Good movement result. The swelling of the glands subsided.

Microscopic examination confirmed the diagnosis of lymphangioma.

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